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EPA (Environmental Protection Agency) Indoor Air Quality Implementation Plan Appendix A. Preliminary Indoor Air Pollution Information Assessment

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16. ABSTRACT

The "Preliminary Indoor Air Pollution Information Assessment" summarizes and assesses information from the published scientific literature regarding sources of indoor pollutants, monitoring methodology and instrumentation, exposure, health effects and mitigation strategies. Information is pollutant specific, and where possible, source specific. Gaps in scientific information are identified to form the basis of a research needs statement addressing specific research initiatives needed to fill these gaps.

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EPA Indoor Air Quality Implementation Plan

Appendix A: Preliminary Indoor Air Pollution Information Assessment

U.S. Environmental Protection Agency
Office of Research and Development
Office of Health and Environmental Assessment
Environmental Criteria and Assessment Office
Research Triangle Park NC 27711

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PRELIMINARY INDOOR AIR INFORMATION ASSESSMENT

PREFACE

This document has been prepared by the U.S. Environmental Protection Agency's Office of Research and Development (ORD) to serve as the scientific basis for the development of a coordinated multi-disciplinary research program which focuses on risk reduction from indoor air pollution. The Agency's Science Advisory Board (SAB), in reviewing the Agency's research program in August, 1986, concurred that such an assessment would be useful in identifying gaps in knowledge, methodology or analysis, which can be filled by research. The purpose of the document is to assemble and assess the world's scientific literature on indoor air pollution, and to summarize this information in a manner that permits identification of knowledge gaps that need to be addressed through research. A further purpose is to contribute to an analysis of indoor air problems which will assist in formulating an effective Agency policy aimed at reducing public health risk by reducing exposure to indoor pollutants.

This iteration of the preliminary assessment has been produced in a narrow time-frame, from October, 1986 through April, 1987. Contributors from offices and laboratories throughout ORD have participated in its writing and review. An earlier iteration was reviewed by a group of twenty expert scientists in indoor air assembled by Harvard in January, 1986, and was reformatted and refined, in the light of expert opinion, and the availability of new information into the present document. One of the issues specifically remaining to be addressed is the problem of determining exposure to indoor pollutants, which would permit a clearer estimation of the risk that various pollutants, or pollutant mixtures, pose to the public. A workshop on the problem of estimating exposure to indoor air contaminants is planned as part of this process. The assessment will also be available to public scrutiny and review. A public information document which draws on the information in this assessment, is also projected.

The present preliminary assessment is divided into an introductory chapter, which includes a history of the indoor air efforts of the Agency and a profile of the ongoing evolution of policy, and four chapters, summarized below.

Pollutant category chapter	• Summarizes scientific information on a chemical-specific basis
Building systems chapter	• Addresses indoor problems across building-related parameters
Welfare effects chapter	• Addresses economic aesthetic and other effects not directly related to health
Source category chapter	• Summarizes pollutant information by source, and leads into research strategy and plan, which for practical reasons is organized by source, as well as by generic concerns spanning several sources.

1. INTRODUCTION

1.1 HISTORICAL BACKGROUND

When the Clean Air Act was passed in 1970, the air pollution problems of greatest concern to the nation were out-of-doors. The "mounting dangers to the public health and welfare," as described by the Congress in Title I, were perceived to be caused by "urbanization, industrial development, and the increasing use of motor vehicles...." Consequently, the law that was intended to protect and enhance the quality of the nation's air resources gave the U.S. Environmental Protection Agency (EPA) authority to control a wide variety of air emissions sources and air pollutants that contributed to the degradation of ambient air. EPA interpreted the term "ambient" to apply to outdoor air only.

The quality of indoor air was not mentioned in the law. At that time, except for studies of specialized environments like submarines, space cabins, and the industrial workplace, virtually no scientific research had been done on indoor air quality. Indoor air pollution and its associated health effects were considered neither serious enough nor pervasive enough to merit national attention.

However, in the early 1970s, indoor air pollution received increasing public attention when the Government instituted energy conservation measures. During this time, formaldehyde was identified as the cause of acute irritant reactions, primarily eye and nose irritation and respiratory distress, in individuals living in homes insulated with urea formaldehyde foam insulation (UFFI), and mobile homes constructed with large quantities of particleboard and plywood. This led to additional research to assess the types and quantities of air pollutants found in various indoor environments, all of which came to the same conclusion: pollutant concentrations were often much higher indoors than they were outdoors. Furthermore, when high exposure levels were coupled with the fact that most people spend more of their time indoors than outdoors, the risk to human health from indoor air pollution was shown to be potentially greater than the risk posed outdoors.

As the general problem of indoor air pollution was drawing more and more public attention as a potential health hazard nationwide, a particular type of indoor air pollutant, radon, was causing immediate concern in certain parts of the country. Epidemiological studies of underground miners had established a link between exposure to elevated levels of radon and the development of lung cancer. In the late 1960s and early 1970s, EPA had investigated homes in Grand Junction, Colorado, contaminated by uranium mill tailings, a by-product of uranium mining. The elevated radon levels found in those homes led to the issuance of the Surgeon General's guidelines regarding remedial action in houses built on or with uranium mill tailings.

During the 1970s, EPA also investigated instances of elevated radon levels in houses built on reclaimed phosphate mines in central Florida. In 1979, EPA issued guidelines to the State of Florida for remedial action in existing homes and for new home construction. In 1983, the Agency began to clean up, under the Superfund program, a number of homes in New Jersey that were built on industrial radium wastes sites.

National attention was focused on the problem of indoor radon in 1984 when a worker at a nuclear power plant in Pennsylvania was found to be living in a house that was contaminated by extremely high levels of radon. In this case the radon was being emitted by the natural soil on which the house was built. Subsequent investigations revealed that thousands of homes in the Reading Prong, a geological formation that runs from Pennsylvania through New Jersey and into New York, were contaminated by naturally occurring radon. Public concern over the potential health effects of radon exposure, and the realization that such exposures could be occurring over wide areas, led to the establishment of EPA's Radon Action Program, directed specifically at the indoor air pollution problem caused by radon.

In fiscal years (FY) 1982 and 1983, the Congress appropriated $500 thousand each year specifically for EPA to conduct research on indoor air quality. Eventually, a total of approximately $7 million was appropriated--roughly $2 million per year in FY 1984, FY 1985, and FY 1986. Of the monies spent in FY 1984 and FY 1985, approximately $300,000 was spent to conduct research on mitigation technologies for radon. In FY 1986, $1.5 million, in addition to the $2 million for research on indoor air pollution, was appropriated for radon research. The resources set aside for radon research were supplemented by $200,000 in FY 1984, $200,000 in FY 1985, and $3 million in FY 1986 for program activities associated with radon.

EPA's indoor air research program has recently been reviewed by the Agency's Science Advisory Board (SAB). In August 1986, EPA asked the Indoor Air Quality Research Review Panel (IAQRRP) of the SAB to review the Agency proposal for assessing the current state of knowledge of indoor air pollution, and to review the relevance of ongoing and proposed Agency research projects related to indoor air pollution.

The SAB concluded that the indoor air research program being conducted by EPA was of high quality and had contributed to the overall understanding of indoor air pollution. However, the SAB felt that EPA's program at the time of the review was lacking a clearly stated policy objective and a clearly focused program for research support. The SAB recommended that as a preliminary planning step, EPA rapidly conduct an assessment of available information and prepare a research strategy and plan based on that assessment.

1.2 THE EMERGENCE OF AN INDOOR AIR PROGRAM AND POLICY

Subsequent to the SAB review of EPA's Indoor Air Program, Title IV of the recently passed Superfund Bill (PL-99-499), the Radon Gas and Indoor Air Quality Research Act (RGIAQRA), came to provide clearer Congressional direction for EPA's indoor air program.

Section 403 of the Superfund legislation directs in part that the Administrator of the EPA establish an indoor air quality research program designed to contribute to the understanding of health problems associated with indoor air pollutants. Also, the statute directs that EPA coordinate with Federal, State, local, and private sector research and development efforts related to improvement of indoor air quality and assess appropriate Federal actions to mitigate environmental and health risks associated with indoor air quality problems. Section 403 of the statute encourages EPA to disseminate information regarding indoor pollutant sources, strengths, and concentrations, high-risk building types, measurement instruments, health effects, as well as recommended methods for the prevention and abatement of indoor air pollution.

As recently stated by EPA to Congress, in response to the new legislation, the goals of EPA's indoor air quality program will be to:

- identify the nature and magnitude of the health and welfare problems posed by indoor air pollution; and

- reduce the risk to human health and productivity from exposure to indoor air pollution.

EPA's program to achieve these goals is based on the belief that non-regulatory programs (i.e., research and development, surveys and assessments, information dissemination, technical assistance, and training) are the most appropriate mechanisms for responding to the problem.

Due to the complexity of the issue, EPA is beginning to adopt a dual-program approach. Efforts will focus both on the sources of individual pollutants and products found indoors, and on the various building types in which pollutants are found. Indoor air pollution can be prevented or mitigated by reductions in levels of specific pollutants emitted by specific products. Traditionally, improvements in environmental quality have been achieved by controlling these specific pollutants or sources of pollutants. However, there is another facet to indoor air quality. Indoor air pollution also occurs as a function of the ways in which buildings are designed, operated, and used. Thus, a second way to reduce indoor air pollution is to approach it as a buildings problem and to change the ways in which buildings function. EPA's indoor air quality program will attempt to address the problem from both perspectives.

The Agency's indoor air research program will be run by EPA's Office of Research and Development (ORD). The Office of Air and Radiation (OAR) will implement a program to develop and disseminate information and guidance to State and local governments, private sector organizations, and the public.

1.3 PRELIMINARY INFORMATION ASSESSMENT AND RESEARCH NEEDS STATEMENT

The EPA's ORD produced a draft document which was a preliminary assessment of available indoor air pollution literature. This draft comprised a synthesis of available information in the form of a summary document, which responded to the concensus of both Congress and the SAB. At EPA's request, Harvard University organized a workshop with 20 of the world's leading researchers in indoor air pollution, rapporteurs from Harvard's faculty, and twelve EPA researchers as observers. The purpose of this workshop was to examine the draft assessment critically for gaps in information, and for the methodology of approach. Thoughtful and constructive suggestions from the workshop participants initiated a change in the format of the presentation of information. The new format presents pollutant categories and examines each from the standpoint of characterization, occurrence and sources, monitoring, exposure, health effects, and mitigation. A summary delineates the important knowns and unknowns for each pollutant category, which then point to research needs. Participants in the Harvard workshop also suggested that while specific exposure assessments were at present not possible for most pollutants, crude exposure estimates by source prevalence and use patterns were an important first step at risk estimation from indoor pollutants. Since building design and ventilation encompass many pollutant categories, as well as mitigation of exposure, workshop participants suggested that it should be discussed in a separate chapter (see Summary of the Harvard Review Attachment A of this document).

The purpose of this document is to assemble and synthesize information on indoor air pollution from work being done by the research community within EPA and other agencies, State and local governments, universities, research institutions, and individual scientists throughout the world. The objective of this volume is to give an overview of available information and to provide an assessment that is intended to serve a number of purposes. Primary among these purposes are 1) recognition of the multidisciplinary nature of the indoor problem, and 2) an identification of research needs for responsible Government agencies and the scientific community at large. With the integration of exposure and health effects data, indoor air problems can be prioritized. Integration of effort among the engineering, monitoring, and health effects laboratories can then provide a research strategy to address specific concerns, while redundancies of effort become evident.

Through this assessment EPA can begin to identify those questions which, when answered by research results, will allow EPA and other Federal agencies to better educate the public about available options regarding indoor air quality. Individual homeowners as well as designers, builders, manufacturers, and maintenance professionals will be made aware of the sources of contamination, as well as the relative hazard that indoor pollution may represent. Other benefits of this assessment, and the resulting research strategy, include new methods of assessing problems, the rethinking and refinement of monitoring techniques, the definition of health parameters affected, and the identification of specific populations at risk. Public awareness of potential hazards associated with improper use and storage of chemicals (which can pollute indoor spaces) for example, must be increased. Means to avoid or mitigate potential problems can be described, so that affected persons can make intelligent choices regarding the contaminants to which they are exposed. State and local health organizations can play a vital role in disseminating information packages developed by the Agency which will be the end product of the new policy on indoor air quality.

This Volume is organized so as to begin answering the following questions:

1. What pollutants exist in indoor environments? (Pollutant Characterization; Monitoring)
2. Where are they? (Characterization of Indoor Spaces)
3. Where do they come from? (Sources)
4. What is the human exposure to indoor pollutants? (Exposure)
5. What are the effects on the human receptor? (Health Effects)
6. Who is affected? (Health Effects)
7. What are the health hazards? (Health Effects)
8. What are the welfare concerns? (Welfare Concerns)
9. How can exposure be mitigated? (Mitigation)

The preliminary assessment presented here will undergo review by experts in indoor air quality and technical experts within EPA and other agencies of the Federal Government. It will continue to be revised in accordance with these reviews and, after completion, will be made available to the general public for examination, by Federal Register notice, prior to a public review workshop. In that this information on indoor air pollution is of interest and importance to a broad audience, EPA plans to ensure that it will continue to be reviewed and revised in an open public forum as new information and analyses on indoor air pollution become available.

1.4 SUMMARY OF WHAT IS KNOWN ABOUT INDOOR AIR POLLUTANTS

Indoor pollutants can be grouped into categories and the information regarding them summarized in relatively brief terms. For a number of indoor air contaminants the source is known, while for some it is surmised. Health effects are also known for some specific pollutants, from human clinical, epidemiologic or animal studies focused on occupational or ambient air situations. For certain classes of compounds only general effects are known and dose-response parameters have not been determined. Specific monitoring information

information for most indoor pollutants is still relatively sparse, although active and some passive monitoring methods have been developed for some specific pollutants or pollutant classes. Development of risk estimates for specific pollutants is not feasible presently, and may not be of practical value. This is especially true for high risk populations, whose responses to indoor pollutants must be extrapolated from human clinical, epidemiologic or chamber studies. Almost no information is available about the interactions of the various indoor pollutants, either chemically, in the mixture that individuals breathe, or their additive or synergistic effects within the body. Estimation of health hazards from specific indoor situations or sources may be a more useful approach than trying to derive risk estimates since it can give the inhabitant of the indoor space options to choose in mitigation of hazard and promotion of healthful environments.

Combustion products can be divided into combustion gases, principally nitrogen dioxide, carbon monoxide and sulfur dioxide, combustion particles, and combustion organics, including PAH and formaldehyde. The indoor sources for all of these contaminants are either combustion appliances such as gas stoves, water heaters, dryers or gas or kerosene space heaters or environmental tobacco smoke (ETS). Fairly specific information is available concerning the three major combustion gases, less is known regarding particles, especially about their interaction with combustion organics.

While NO_2 is usually monitored passively over a one to two week period, it can be monitored by this mode for as little an 8-hour day. Active monitoring is typically done hourly, but can be done in real time. This is important since health effects appear to be more dependent on short-term exposure to high levels of NO_2, rather than by chronic, low level exposures. Personal exposure monitoring is also possible, which together with diaries detailing personal activity, can pinpoint high-level exposure peaks more accurately. Chamber studies indicate that exposure to concentrations greater than 0.3 ppm NO_2 for 15 minutes can decrease pulmonary function in asthmatics, while levels greater than 2.5 ppm for longer periods of time increase susceptibility to infection in animals. Exposure to between 1 to 2.5 ppm has been shown to decrease pulmonary function in children and perhaps adults. Evidence from animal studies, and some epidemiological studies, indicate an interactive response with other pollutants, resulting in decreased immune response and changes in anatomy and function of the lung. Actual measurements of NO_2 during cooking with gas or during use of kerosene heaters have shown levels exceeding 1 mg/m^3 (0.53 ppm). Census data show that 95,802,000 people cook with gas stoves averaging 4 hours per day, while 7,022,000 individuals are exposed to kerosene heater fumes averaging 2 hours per day. These numbers allow a generalized estimate of the potential hazard from NO_2 to those people exposed, but lack of dose-response and specific concentrations and exposure times, as well as variation in susceptibility do not permit a risk estimate for any of the health effects.

Carbon monoxide is monitored presently by use of personal exposure monitors which together with activity diaries can yield good estimates of personal exposure. Relatively few studies exist which have monitored CO concentrations and exposure times, however. The existing studies indicate CO exposure in vehicles (cars) in commuter traffic, as well as from ETS and faulty or unvented combustion appliances. The extent of population exposure to the latter is not known, since symptoms of moderate to high level concentrations mimic many illnesses, and CO poisoning symptoms are largely not attributed to CO, but to illness such as flu, or food poisoning.

CO attaches avidly to hemoglobin in the human bloodstream, forming compound carboxyhemoglobin (COHb); exposure to CO can be measured as the percent of hemoglobin which is in the COHb form. COHb cannot transport oxygen to tissues, and tissues with high oxygen demand, such as the brain and heart, are most rapidly affected by the resulting oxygen deprivation. At 2.9 to 4.5 percent COHb patients with angina pectoris have an aggravation of their attacks in frequency and duration. At levels above 5 percent COHb, healthy males show a decrease in work capacity, while at 10 percent COHb patients whose cardiopulmonary function is compromised show an exacerbation of their dysfunction. Between 10 to 30 percent COHb, healthy adults show decreased mental alertness, suffer headaches and exibit flu-like symptoms. At COHb levels greater than 60 percent, asphyxiation occurs. If death does not follow, permanent brain damage from anoxia can result.

Monitored concentrations of CO relate to percent COHb through a relationship developed by Coburn et al. (1965), which permits calculation of blood COHb as a function of time, considering appropriate physiologic and physical factors. Up to 60 ppm CO have been measured inside cars stopped in traffic jams, while up to 18 ppm were measured in public garages. With faulty combustion appliances concentrations in excess of 100 to 200 ppm have been measured.

There is presently no way of determining the number of people living in "sick" homes with faulty or unvented combustion appliances. Even when properly vented, homes that are very weather-tight may have down-drafts through the chimney which can cause dangerous levels of CO (and NO_2).

Sulfur dioxide is also a combustion gas, but is derived from burning fuels with high sulfur content such as some natural gas and kerosene or oil. It is monitored passively, usually measured over 12 hours, and not actively monitored indoors.

Chamber studies have shown that concentrations of less than 0.75 ppm administered for less than one minute have caused a decrease in lung function in infants and the elderly, in synergism with inhaled particles. Lung airway resistance doubled in the affected individuals at this exposure. Monitored concentrations with use of low-sulfur fuel in unvented kerosene heaters has determined concentrations of 0.1 to 2.0 ppm/12 hour average. Acid aerosol emissions from kerosene heater use can be extremely high. The number of people exposed to SO_2 indoors and the extent of their exposure is not known at present.

Inhalable particles are produced in cigarette smoke and by combustion appliances. At present there are no indoor passive monitors for particles, but there are active fixed and personal monitors which may be used indoors. Obtaining adequate sample sizes with such monitors is difficult, but larger samplers or larger-volume samplers are often too bulky and obtrusive for indoor use.

Cancer is the most serious health consequence that has been associated with inhalable particles. Soot itself is carcinogenic, and pro-, co- and frankly carcinogenic polycyclic aromatic hydrocarbons are adsorbed to fine particles which are inhaled deeply into the lungs where they can deliver their dose. Particles themselves may play a synergistic or additive effect in the carcinogenesis of these adsorbed compounds. Composition of these entities varies with fuel and burning conditions and no dose-response information is known. Another effect is irritation of respiratory tissue and the eyes, at variable concentration, depending in part on particle composition. Decrease in lung function occurs at concentrations of respirable particles greater than 300 $\mu g/m^3$, alone, or in synergism with SO_2 or other gases.

Monitored peak concentrations from combustion appliances indoors have reached 119 $\mu g/m^3$. Concentrations of particles in ETS can increase this value several-fold. While the aggregate exposure to particles from combustion appliances is not known, it has been calculated that 86 percent of nonsmokers exposed to ETS (124,700,000 people) experience a peak exposure of 1.43 mg/day.

Many organic products of incomplete combustion enter the indoor environment from cigarette smoke and from unvented combustion appliances. Only some of these organics have been identified, fewer have been quantified. A large number of PAH have been measured by active 8 hour-average monitoring. One to 3 ng/m^3 large molecules and 20 to 50 ng small molecules have been collected over this time period from ETS. However, dose-response information is not known. Among the health effects determined in epidemiologic studies are cancers, cardiovascular effects and irritation of mucous membranes. Animal data show a decrease in immune function with exposure, and a role in the etiology of atherosclerosis. Exposure to combustion organics from unvented or faulty appliances is not known, and exposure to ETS is included in the discussion on particles.

Formaldehyde is a combustion organic that is also a compound released from sources such as building material and furnishings. It will be discussed under the gas-phase organics. Its primary source under combustion organics is ETS and its primary effect is cancer, with irritation of mucous membranes and allergic reactions less lethal but more common.

Among the noncombustion particles of concern in indoor air are asbestos, dusts, sprays, and cooking aerosols. Biologic particles will be discussed separately.

Asbestos fibers derive from asbestos cement and insulation used in some residences, schools and public buildings built prior to 1970. It was used to insulate steam-boilers and steam pipes. As this insulation ages it can become friable, and fibers can be entrained into breathable air. Vehicles can also be a source of asbestos from wear on brake linings. Since asbestos fibers fall into the particle classification, they can be collected by active samplers and by PEM, but require special electron microscope analysis for fiber counts. Little measurement has been done to determine extent of exposure, except in a few public buildings, and in schools. Monitoring performed in schools throughout the U.S. can give ranges of concentrations found (20 to 4500 ng/m^3; approximately 0.0006 to 0.15 fibers/mL, ±4 f/mL). The number of children potentially exposed for 6 hours per day in older schools is 19,783,000. Approximately 33,387,000 people are potentially exposed to asbestos in steam-heated homes built prior to 1970 for 10 to 24 hours per day. Commuters in cars and buses, who number about 217,500,000 and at least an additional 38,100,000 truck drivers, are exposed to asbestos fibers from brake linings.

Dusts, sprays and cooking aerosols result from personal activities and vary in their concentrations depending on the duration and extent of such activity. Practically no monitoring efforts have been made to distinguish and quantify these entities and methods development is still needed. Since the identity of the components of such mixtures is not known, neither exposure nor health effects data are available.

Noncombustion gas-phase organics (also known as VOCs or volatile organic compounds. This is a misnomer since many have rather high boiling points and are not very volatile; many semi-volatile organic compounds are included in the designation) constitute a broad class of compounds of varying reactivities and physical properties. Their sources are paints, stains, adhesives, dyes, solvents, caulks, cleaners, pesticides, and building materials and furnishings found in homes, offices, public buildings, and vehicles. No passive monitoring is feasible for these gas-phase organics but active monitors for indoor sampling, usually for 24-hour averages, are available, as are PEMs. More than 900 different compounds have been identified in indoor environments and many more remain unidentified. Health effects for some individual compounds are known, but concentrations at which such identified health effects have been noted have been much greater than those generally measured in indoor air. VOCs occur in mixtures, however, whose aggregate effect is not known. However, many gas-phase organics have been measured indoors at concentrations greater than that in the ambient air, where the concentrations are regulated. Effects range from sensory irritation to behavioral and neurotoxic effects, hepatotoxic effects, and cancer. No epidemiologic work has been performed to relate concentrations with such health effects. Concentration-response relationships are known for individual compounds, and cancer risk numbers have been calculated for some compounds by the Carcinogen Assessment Group of the U.S. EPA. Concentration-response effects for aggregate mixtures of commonly found gas-phase organics in office buildings in Denmark have been determined in chamber studies, with 5 to 25 mg/m^3 causing synergistic behavioral changes in sensitive adults. However, no health effects data is available for assessing the effect on neurologic, liver functions, respiratory system changes or for the risk of various cancers from the effects of aggregate mixtures of gas-phase organic compounds. Estimates of exposure may be possible for individual compounds such as formaldehyde, in specific indoor settings, but because of the multiple sources, the ubiquitousness of the compounds, and the lack of monitoring data generally, no real estimate of exposure is possible at this time.

Formaldehyde (HCHO) is a gas-phase organic compound that is a combustion product but also is emitted from urea formaldehyde foam insulation (UFFI), resins from particle board and plywood, furnishings, carpets and upholstery. Because of very high concentrations emitted from UFFI, considerable attention has been focused on this contaminant, and it has been identified as a problem in certain environments, especially in manufactured homes, due to the prevalence of sources there. Passive monitors are available. These required 5 to 7 days monitoring time. Both active stationary samplers requiring two hours averaging times, and PEM are available. Health effects such as irritation of mucous membranes have been shown to occur in chamber studies at 0.1 to 0.2 ppm. Individuals sensitized to HCHO react allergically at concentrations of less than 0.1 ppm, however. EPA has recently reclassified HCHO as a possible human carcinogen, based largely on data from animal studies. Concentrations measured in mobile homes range from 0.03 to 8 ppm, with 9,279,000 people potentially exposed in these environments for 10 to 24 hours per day. Concentrations in offices, public buildings, schools and homes can also range into irritatory levels, especially after remodeling or after installation of new furnishings and carpets. Formaldehyde is a suspected actor in sick building syndrome. The extent of exposure in environments other than mobile homes has not been estimated.

Radon emanates from radium-containing soil and enters residences through cracks or other openings in basements, or through crawl spaces. It is passively monitored. Concentrations measured in various parts of the U.S. have typically ranged from 0.5 to 2×10^3 pCi/L. The U.S. EPA considers a level of 4 pCi/L to be the action level at which mitigation should occur. An estimated 93,760,000 people in the U.S. live in states with an average radon concentration greater than 4 pCi/L and are exposed to levels at or greater than this. The U.S. EPA has published risk estimates which predict 5,000 to 20,000 excess cancers/year for exposures presently projected.

Biological contaminants represent a very diverse group of substances in indoor environments. They include infective agents such as viruses, bacteria and molds, capable of causing infectious diseases. Some bacteria and molds produce highly toxic substances which have effects on many systems in the human body, and thereby constitute a threat aside from virulence. In addition these viable agents can cause allergic reactions, as can mold and bacterial spores, pollen, insect, acarid, and arachnid body parts and excreta, and animal and human dander. While a variety of passive and active methods for monitoring biological contaminants exist, passive monitors do not collect representative samples, and active monitoring methods have not been standardized. Without standardized methodology, results from different researchers are not comparable. Baseline data to distinguish normal levels from contamination are not available. Since a clear distinction of what constitutes contamination by the various biologic agents has not been made, the extent of exposure cannot be estimated.

Pesticides are organic chemicals applied in and around buildings, primarily to control insects. They can enter indoor spaces through cracks and other openings in foundations, or be directly introduced. They are by definition poisonous substances, affecting the nervous system, the liver, or the reproductive systems. Allergic reactions have been documented. Dose-response relationships for toxicities for individual pesticides are known in some cases from industrial and animal studies. Since they are organic substances, primarily in vapor-phase, they are monitored actively by the same methodology as other gas-phase organic compounds, specifically semi-volatile organic compounds. Concentrations measured through the NOPES program have detected aggregate concentrations in the range of 1.7 to 15 $\mu g/m^3$. Cumulative effects of different pesticides and interactions with other organic compounds have not been determined. Monitoring has not been extensive so that exposure estimates for pesticides are not possible at this time.

Nonionizing radiation is produced by all electric conductors which generate electric and magnetic fields around themselves whenever a current flows through them. Such conductors are ubiquitous. Animal and epidemiologic studies have shown behavioral and reproductive effects, and possible cancers resulting from the influence of low level electric and magnetic fields. The dose-response is not known. Such fields can be detected by passive monitors. Field strengths measured are in the range of 1 to 2 m gauss for magnetic fields and 10 mV/m^2 for electric fields. All individuals who use electricity or come near electric conductors are exposed to such nonionizing radiation, but the effects of such exposure on the human population is unknown.

Environmental tobacco smoke is a complex mixture of pollutants whose source is primarily cigarette smoking. Various components of this mixture have been actively monitored and include respirable suspended particles, CO, nicotine, nitrogen oxides, acrolein, nitroso-compounds and benzo(a)pyrene. Health effects from ETS have been extensively discussed in both the 1987

Surgeon General's report and the 1986 report from the National Academy of Sciences. They include cancer, cardiovascular effects, increased susceptibility to infectious diseases, chronic and acute pulmonary effects in children, mucous membrane irritation and allergic response. Approximately 124,000,000 people are exposed to up to 1.43 µg/day of ETS per day. Dose-response effects are not known.

Since dose-response effects for indoor pollutants and even concentration-response effects are generally not known, it should be emphasized that risk estimates for populations are not presently possible. This is espeically true since individuals are exposed to a wide variety of complex mixtures of pollutants whose interactive risks are unknown. It is possible to state numbers of people potentially exposed to an indoor situation or a source that poses a hazard, and to suggest ways to mitigate the source or the potential exposure for people. Individuals can then be presented with options for avoiding hazards or choosing or avoiding activities or sources that impinge on their health.

Table 1-1 summarizes the information described in this narrative in abbreviated form.

TABLE 1-1. SUMMARY: WHAT IS KNOWN ABOUT INDOOR POLLUTANTS

Pollutant	Source	Health Effect	Concentration for Effect	Monitoring Indoors			Concentration Measured/ Activity	Estimated Number of People Exposed	Estimated Length of Exposure/ Day
				Passive/ Time	Active/ Time	PEM			
NO_2	Combustion Appliances ETS	Decreased pulmonary function in asthmatics	1 ppm NO_2 15 min	1 day (8 hr) (minimum)	1 hr	Yes	Up to 1 mg/m^3 during cookinf with gas	95,802,000	4 hrs
		Increased susceptibility to infection	>2.5 ppm NO_2	1-2 weeks	Real time		Up to 1 mg/m^3 use of kerosene heater	7,022,000	2 hrs
		Effect on pulmonary function in children, perhaps adults	1-2.5 ppm NO_2						
		Synergistic detrimental effects with other pollutants	Varies						
		Animal studies indicate decreased immune capability, changes in anatomy and function of lung							
CO	Combustion Appliances ETS Infiltrated auto exhaust	Aggravation of angina in patients	2.9-45.% COHb	No	Yes, for outdoors; too bulky indoors	Yes	Up to 18 ppm in public garages Up to 60 ppm in vehicles in traffic jams	Unknown	Unknown
		Decreased work capacity in healthy adult males	5% COHb				100-200 ppm with faulty appliances, no venting	Unknown	Unknown
		Headaches, decreased alertness, flu-like symptoms	10-30% COHb						
		Exacerbation of cardiopulmonary dysfunction in compromised patients	10% COHb						
		Asphyxiation	>60% COHb						

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TABLE 1-1. (continued)

Pollutant	Source	Health Effect	Concentration for Effect	Monitoring Indoors Passive/ Time	 Active/ Time	 PEM	Concentration Measured/ Activity	Estimated Number of People Exposed	Estimated Length of Exposure/ Day
SO_2	Combustion of fuels containing sulfur (e.g., kerosene, oil	Decreased lung function in infants, elderly (in synergism with particles) increased (doubled) airway resistance	(0.75 ppm/ 1 min)	Yes	Yes	No	0.1 to 2.0 ppm/ 12 hr average with unvented kerosene heater use, low sulfur fuel	Unknown	Unknown
		Animal studies show decreased lung function	1 ppm						
Combustion particles	Combustion Appliances ETS	Cancer (soot, PAH absorbed to particles)	Dose-response unknown	No	Yes	Yes	119 μg/m³	Aggregate combustion appliance exposure unknown	1-10 hrs
		Irritation or respiratory tissues, eyes	Varies					ETS: 86% of non-smokers exposed (124,700,000 at 1.43 mg/day, peak	
		Decreased lung function, alone and synergistically with SO_2, other gases	>300 μg/m³						
Combustion organics:	Combustion Appliances							Aggregate combustion appliance exposure unknown	Unknown
PAH's	ETS Kerosene Heaters	Cancer, irritation, cardiovascular effects	Dose-response unknown	No	Yes 8 hr	Yes	1-3 ng/m³, large molecules	124,000,000 exposed to 1.43 mg/day ETS residue	2-10 hrs
		Animal data show decreased immune function, atherosclerosis etiology					20-50 ng/m³, small molecules		

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TABLE 1-1. (continued)

Pollutant	Source	Health Effect	Concentration for Effect	Monitoring Indoors			Concentration Measured/ Activity	Estimated Number of People Exposed	Estimated Length of Exposure/ Day
				Passive/ Time	Active/ Time	PEM			
Combustion organics: Formaldehyde (see below)	Combustion Appliances	Irritation							
	ETS	Cancer							
Noncombustion particles									
Asbestos	Asbestos cement, insulation Floor tiles Brake linings	2 fibers/m^3/50 yrs		No	Yes	Yes			
Dusts Sprays Cooking aerosols	Personal activity	Unknown; can range from irritation to cancer	Unknown	No	No	No	Unknown	Unknown	Unknown
Noncombustion gas-phase organics (VOC)	Paints Stains Adhesives Dyes Solvents	Irritation Neurotoxic/Behavior effects	Varies Dose-response to aggregate VOCs: 5-25 mg/m^3	No	Yes 24 hrs	Yes	Individual compounds		
	Caulks Cleaners Pesticides	Hepatotoxic effects	Dose-response known for individual organics						
	Building materials	Cancer	Risk estimated for individual compounds						
Formaldehyde	UFFI Particle board	Irritation	0.1-2.0 ppm	Yes 5-7 days	Yes 2 ppb-hrs	Yes	0.03-8 ppm in mobile homes	9,279,000 in mobile homes	10-24 hrs
	Plywood	Allergy	>0.1 ppm						
	Furnishings Carpets Upholstery	Cancer	Dose-response unknown						

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TABLE 1-1. (continued)

Pollutant	Source	Health Effect	Concentration for Effect	Monitoring Indoors Passive/ Time	Active/ Time	PEM	Concentration Measured/ Activity	Estimated Number of People Exposed	Estimated Length of Exposure/ Day
Radon	Soil Well Water Some building materials	Cancer	Indoor dose-response unknown	Yes	No	No	0.5-2 x 10^6 PCi/liter	2,500,000	10-24 hrs
Biological contaminants: Viruses Bacteria Molds Insect and arachnid excreta Pollen Animal and human dander	Outdoors Humans Animals Moist building areas	Infectious diseases Allergy Intoxication	Varies; mostly unknown	Yes (useless)	Yes (not standardized)	No	Varies	Unknown	10-24 hrs
Pesticides	Outdoors Indoor spraying Pets Moth control	Neurotoxicity Hepatotoxicity Reproductive effects	Dose-response for individual pesticides is known	No	Yes	Yes	Varies 1.7-15 $\mu g/m^3$	Unknown	10-24 hrs
Nonionizing radiation Electric and magnetic fields	Electric conductors	Possible cancer, behavioral and reproductive effects	Dose-response unknown	Yes	No	No	1-2 mgauss 10 mV/m^3	Unknown	10-24 hrs
ETS	Cigarette smoking	Cancer	Dose-response unknown	Some components: particles, CO, nicotine, NO_x acrolein nitro-compounds Benzo-a-pyrene quantified			124,000,000 exposed to 1.43 mg/day	2-10 hr	
		Irritation to mucous membranes	Varies						
		Chronic and acute pulmonary effects in children	Dose-response unknown						
		Cardiovascular effects	Dose-response unknown						

2. POLLUTANT CATEGORIES

2.1 INTRODUCTION

Individuals breathing the air within indoor spaces are exposed to complex mixtures of pollutants whose total impact on health and well-being is presently not known. The interactions that occur chemically and physically among the various pollutants are not well characterized; their combined interactions on and with materials found indoors are also not well understood. Their synergistic, antagonistic, and additive effects on health parameters are not known. Pollutants are often identified with a specific source, that is, a gas stove, and individual pollutants from such a source are measured and correlated with source presence or use, but other emission components are not measured. Most of the information relating to exposure and health, especially that coming from animal or clinical studies, derives from investigation of single pollutants. For these reasons the information known about indoor pollutants is here organized into single pollutant categories; when a complex mixture such as environmental tobacco smoke (ETS) has been studied as a source entity, it is presented as such.

The chapter is organized to give an overview of published information about selected pollutant categories as follows: combustion gases, including carbon monoxide (CO), nitrogen dioxide (NO_2), and sulfur dioxide (SO_2); particles and combustion organics, including gas phase organics and polycyclic aromatic hydrocarbons (PAH), among others; ETS; noncombustion particles such as asbestos fibers, dust, spray and cooking aerosols; noncombustion gas-phase organic compounds (also called volatile organic compounds or VOCs); radon; biological contaminants; pesticides; and nonionizing radiation. For each of these pollutant categories, if such information is available, the chapter discusses occurrence or sources, estimates of exposure, including exposure levels and distributions among the population, and a brief discussion of monitoring techniques and the factors that affect monitoring, such as averaging times. Health effects, which are the driving force for a consideration of indoor pollutants, are summarized next, including results from in vitro, animal and human studies, concentration-response relationships where known, and the length of exposure known to produce effects.

Since the goal of the Indoor Air Quality program is to reduce exposure for the total population, this analysis is followed with a discussion of mitigation and control options for each pollutant category. Finally, the information for each pollutant category is summarized to give an indication of the major known and unknown areas of information, which summary leads to an indication of what the needs for future research will be.

Accurate estimation of human exposure is essential for determination of risk posed by pollutants, as well as for the design and implementation of effective exposure reduction methods. In order to determine exposure in a useful way, a clear understanding of the meaning of the term, and related terms, is needed. A distinction must be made among the terms concentration, exposure, and dose. Concentration of an air pollutant is the mass or amount of that material per unit volume of air (e.g., milligram per cubic meter). Monitoring devices measure pollutant concentrations, which may allow accurate exposure estimates to be projected. Nominal exposure is defined here as the contact between the surface of the body (skin, respiratory tract, gastrointestinal tract) and the pollutant. Two events must occur simultaneously to constitute a nominal exposure:

1. A pollutant of a certain concentration is present at a specific location for a measurable time, and
2. A person is present at that location during the given time period. (Duan, 1982; Ott, 1985; Sexton and Ryan, 1987).

The key distinction between a concentration and an exposure is that exposure is defined by the presence of persons in the contaminated environment. Monitored concentrations can act as a surrogate for exposure, on the assumption that they represent concentrations experienced by people. It is an assumption whose utility is especially important for source mitigation considerations, since reduction in concentration of pollutant implies a reduction in exposure.

The distinction between exposure and dose is also important. While exposure is defined as the pollutant concentration at the interface between the body and the external environment, dose is defined as the amount of pollutant that crosses this interface and reaches the target tissue. Factors that affect the size of the dose delivered through the respiratory system are respiration rate, volume, mode (mouth or nose breathing), uptake, metabolism, and clearance. The relationship between exposure and dose has not been established for most air pollutants. The distinction between the terms can be made clear by considering two individuals in the same environment, one at rest, the other exercising vigorously. They both have the same nominal exposure, but clearly the second is receiving a higher dose (Sexton and Ryan, 1987).

With regard to the health consequences of exposure to indoor air pollutants, three parameters of exposure are especially important:

1. Magnitude or pollutant concentration
2. Duration or length of exposure
3. Frequency of exposure.

Magnitude is assumed to be directly proportional to dose, and is an important aspect of exposure, but duration is likewise important. Just as high level or peak exposures are likely to impact the human system differently from low level exposures, the length of exposure (5 minutes versus an hour or a day) is likely to be a determinant of tissue injury. Frequency, too, has an impact, since repeated insult to tissues is likely to do more damage than a single encounter.

Measurement of exposure most often depends on the capabilities of monitoring instruments. Real-time exposure monitors, together with activity diaries, can provide a continuous record of exposure over a time period, and indicate periods of no exposure to a pollutant, as well as frequency, duration and magnitude of exposures. These instruments and the results they provide are often complex, and the data is difficult to analyze. Summaries of data, which average concentrations over time (e.g., average concentration per hour or per day) are more commonly used. Exposure profiles obtained from personal exposure monitors combined with activity pattern diaries give the most complete picture of exposure, including frequency and extent of peaks. Integrated exposures, on the other hand, give a single value obtained from integration of the function ($C_i t$) over a specified time period. This method does not provide information about the pattern and severity of short-term, peak exposures. "Average exposure" is a specialized form of integrated exposure defined as the integral of C_it over the averaging time t_a, divided by t_a.

Individual or personal exposure as measured by time-activity patterns and personal exposure monitors (PEM) can vary substantially from person to person. Estimates of group exposure may require many such individual exposure measurements. From a public health perspective, it is important to know the number and characteristics of the group of people at risk. Such determinations give estimates of "population exposure" or the aggregate exposure for a specified group of people. Since exposures are likely to vary substantially between individuals in a group, the distinction of personal exposures within the group, including the average values, and the variance, should be specified. The upper tail of distribution, which delineates those individuals who experience the highest pollutant concentrations, can be essential to health risk assessment. The lower tail of distribution can identify especially sensitive populations.

At the present time, monitored information which would allow exposure calculations for many indoor pollutants is still too sparse to give good predictions of population exposures. Risk estimates for populations exposed to indoor air pollutants are therefore premature. Information does exist, however, regarding source frequency and usage patterns for many source categories of indoor pollutants. It is therefore possible, at present, to give a crude estimate of the number of people exposed to a range of pollutant concentrations, over differing averaging times. While this does not permit the calculation of risk numbers, it does give a sketch of possible risk that would be helpful towards ranking pollutants by the severity of their impact on the population as a whole. Table 2-1 describes such a profile for selected pollutant sources.

2.2 COMBUSTION GASES

2.2.1 Introduction

Combustion research has demonstrated that the combustion of fossil fuels (e.g., oil, kerosene, coal, natural gas) and vegetative sources (e.g., wood, plant material) can result in the production, and usually the emission, of a very complex mixture of organic and inorganic gaseous and particulate pollutants. In addition, the complex mixture that results from incomplete combustion can contain a variety of other volatile and semivolatile, polar and nonpolar chemical products.

TABLE 2-1. EXPOSURE PROFILE BY SOURCE PREVALENCE AND USE PATTERNS FOR SELECTED POLLUTANTS IN THE U.S.[a]

Source	Pollutants Emitted	Number of People Potentially Exposed	Percent of Total U.S. Population	Estimated Average Number of Hours Exposed Per Day
Combustion Appliances	CO, NO_2, SO_2, PM, HCHO			
	Gas Stoves	95,802,000	41	4
	Gas Water Heaters	100,317,000	43	4
	Gas Dryers	47,023,000	20	1
	Gas Furnaces	115,866,000	49	5
	Gas Space Heaters	20,063,000	8	2
	Kerosene Space Heaters	7,022,000	3	2
	Woodstoves	13,543,000	6	5
	Fireplaces	45,143,000	19	3
Environmental	CO, NO_2, PM, PAHs, HCHO Tobacco Smoke	85,269,000[e]	36	10[f]
Asbestos	Asbestos Fibers (in school buildings)	19,783,000[g]	8	6
Gas-Phase Organic Compounds	Pesticides, HCHO, Solvents, Cleaners	9,279,000[h]	4	10

[a] = Base year: 1983

[b] = Average number of residents per home: 2.5

[c] = 1983 estimated total U.S. population = 234,500,000

[d] = Winter months

[e] = Including smokers

[f] = Estimated average number of hours at home, does not include occupational settings.

[g] = Average number of children per school x number of older schools.

[h] = Estimated number of people living in mobile homes; extent of exposure not known for households, office buildings.

The primary emphasis in this section is on three products of incomplete combustion: carbon monoxide, nitrogen dioxide and sulfur dioxide. The first of these is given off from any incompletely burned carbon compound. Nitrogen dioxide is formed from nitrogen and oxygen in air at high combustion temperatures, and sulfur dioxide is formed during the combustion of sulfur-containing fuels (such as gas or oil). A large body of literature is available on these pollutants since they are classed as regulated criteria pollutants for ambient air. Other gas-phase products of combustion, such as some of the PAHs, are also sources of risk due to their carcinogenicity, but specific information such as dose-response relationships are not known. These are described further in the particle section of this document, since they also adsorb to particle surfaces.

2.2.2 Occurrence and Sources of Combustion Gases

Some typical indoor sources of combustion products and related contaminants are contained in Table 2-2. This table will serve as a basis for the subsequent discussion of indoor combustion sources.

2.2.2.1 Unvented Kerosene Space Heaters. Unvented kerosene heaters can be divided on the basis of flame type into two categories: blue-flame and white flame. The color of the flame is determined by the burner design, the manner in which air enters the burner area, and the flame temperature. Within these two categories there are five basic types of kerosene heaters on the market. They are convective, radiant, radiant/radiant, radiant/convective, and wickless.

The primary pollutants that have been measured from unvented kerosene space heaters are NO_x, CO, carbon dioxide (CO_2), SO_2, and formaldehyde (HCHO) (Consumer Products Safety Commission, 1983). In general, higher flames than normal will increase the emission of NO, while lower flames increase CO, HCHO, and particulate matter (U.S. Department of Energy, 1985). The SO_2 emission rates depend on the sulfur content of the fuel and are not significantly affected by changes in the flame or heater type or age of the unit (U.S. Department of Energy, 1985).

Unvented kerosene space heaters vary widely in their production of indoor pollutants. The variation can be attributed to the wick height, fuel type, adjustment of primary air/fuel ratio, length of time the burner has been operating during a single use period, either over or under firing of the burner, and the overall design of the unit (Consumer Products Safety Commission, 1983; Lionel et al., 1986).

TABLE 2-2. INDOOR AIR SOURCES AND RELATED CONTAMINANTS

Sources	Gases and Vapors	Particles
Unvented appliances	H_2O, CO_2, CO, NO_x, NO_2	large particles fine particles Aitken nuclei
gas-fired		
kerosene-fired	(+)SO_2	
Ventless heaters	H_2O, CO_2, CO	
gas-fired	(+)NO_x, mercaptans	Aitken nuclei fine particles
oil-fired	(+)NO_x, SO_2	large particles
wood-fired	(+)NO_x, SO_2, HC	B(a)P
Automobile garages	CO, HC, nitrogen compounds	fine particles Aitken nuclei

Source: Woods (1983).

Traynor et al. (1983), reported on pollutant emissions from four portable kerosene-fired space heaters. The tests were conducted in a 27 m^3 environmental test chamber operating at an air infiltration rate of approximately 0.4 air changes per hour. The concentrations reported were based on one-hour burn times. The results are summarized in Table 2-3. All four heaters tested were found to emit CO, NO, NO_2, and HCHO. There were no significant differences found in emission rates with or without a warm-up period.

In a subsequent work, Traynor et al. (1984) reported on two kerosene heaters (1 white-flame convective, 1 blue-flame radiant) that were operated in the master bedroom of an unoccupied house. The test attempted to identify the pollutants and to determine interroom transport of those pollutants over a range of test conditions. The test conditions included doors and windows closed; door closed and the window open 2.5 cm; door open 2.5 cm and the window closed; door wide open (74 cm) and the window closed.

In each case the heater was operated until an 8°C increase in temperature was achieved in the room and the increase in CO, CO_2, NO, and NO_2 was recorded. Carbon dioxide concentrations were determined to range from 2440 to 5440 ppm; NO_2 levels ranged from 0.12 to 0.60 ppm. Traynor and co-workers reported interroom transport for those test conditions with the window closed. They

TABLE 2-3. POLLUTANT EMISSION RATES FROM FOUR PORTABLE KEROSENE-FIRED HEATERS

Heater/Fuel Consumption (kJ/hr)	Emission Rates (µg/kJ)					
	CO	CO_2	NO	NO_2	HCHO	Particulate[c]
New Convective						
7830	14.5	70100	23.7	14.1	0.01	<0.004
7980	10.3	72500	25.2	12.5	0.08	<0.004
7840[b]	9.1	70400	25.3	12.2	0.18	<0.004
Old Convective						
5480	115.1	69000	11.1	33.6	1.22	a
5780[b]	110.5	63600	10.9	29.7	0.98	0.006
New Radiant						
8180	60.2	70300	1.4	5.2	0.63	0.019
8250[b]	71.7	68500	1.2	4.1	0.49	0.022
Old Radiant						
6640[b]	54.0	66200	2.1	5.1	0.10	0.019

[a] 440 µg emitted at ignition.

[b] 10-minute warm-up outside chamber.

[c] Particulate values: Mass of particles from 0.005 to 0.4 µm diameter.

Source: Traynor et al. (1983)

found that interroom transport was less than 10 m^3/hr for the pollutant gases with the door closed, 30 ± 10 m^3/hr with the door open 2.5 cm, and ranged from 190 to 3400 m^3/hr with the door fully open (Traynor et al., 1984).

Traynor et al. (1986) have also reported finding that other compounds, including aliphatic hydrocarbons, alcohols, ketones, phthalates, and alkyl benzenes are emitted from kerosene heaters. In a chamber study of convective and radiant kerosene heaters they concluded that the reactivity of semivolatile organics (SVOCs) implies that reactivity rate for SVOCs is more important in determining their indoor concentration than ventilation or air exchange rates. This indicated that further studies are needed to quantify the indoor reactivity processes for individual SVOCs in order to get accurate insight into the exposure (Traynor et al., 1986).

2.2.2.2 Gas Appliances. It has been estimated that 45 percent of American homes use natural gas, and that most do not vent combustion products outside the house (U.S. Department of Energy, 1985). Therefore it is not surprising that the house unvented gas combustion appliances can be a significant source of indoor pollutants. They have been reported to be a source of particulate matter, NO_x, SO_2, HCHO, and CO (Gas Research Institute, 1985; Girman et al., 1982). The factors that appear to affect the pollutant emission rates are usage pattern, burner design and manufacture, age of burner, fuel consumption rate, and combustion efficiency (U.S. Department of Energy, 1985).

2.2.2.2.1 Gas stoves. In homes where gas stoves are used, kitchen pollutant concentrations have been shown to respond rapidly to stove use. For a given house during a given season, there is a rough correlation between average NO_2 concentration and the average stove use. It has been reported that normal stove operation frequently results in NO_2 concentrations in the kitchen that are 100 $\mu g/m^3$ (0.053 ppm) over a 2 week sampling period, with NO and NO_2 being produced in roughly equal amounts (Wade et al., 1975).

Pollutant emissions from newer stoves have not consistently been reported to be higher or lower than those from older stoves. The different designs of the burners do not appear to have a consistent or reproducible affect upon pollutant emissions. An evaluation of the number of burners in use and their flame intensity has shown that emissions are similar for all conditions when adjusted to equivalent heat output (GEOMET Technologies, Inc., 1976).

Oven and broiler emissions were reported to be somewhat less than those of burners on the heat output basis. The total pollutant emissions (per unit of time) from a gas stove are roughly proportional to the number and types of burners and the period of use. Pilot lights appear to contribute quantities of pollutants comparable to those generated during cooking activity over a typical 24-hour period (GEOMET Technologies, Inc., 1976).

Table 2-4 presents emissions rates for gas range and oven (U.S. Department of Energy, 1985).

Two major field studies dealing with the indoor air impact of gas stoves have focused on NO_2. They are the Southern California Gas Corporation study (Colome et al., 1982) of 500 hours (400 with gas stoves, 100 with electric ranges) in the Los Angeles, CA, area and the Harvard-Gas Research Institute study of 600 hours (450 gas, 150 electric) in the Boston, MA area (Soczek et al., 1986).

Both studies included the main components of random selection of a cluster of homes; two-week measurements using Palmes tubes; multiple indoor sampling locations (i.e., kitchen, bedroom, and living room); air exchange measurement; water vapor measurements; and outdoor air measurements.

TABLE 2-4. EMISSION RATES FOR GAS RANGES AND OVENS (mg/hr)

Nonstanding pilot, fuel input rate per burner 8680 to 9060 Btu/hr		
	Rich gas (1022 Btu/SFC)	Lean gas (983 Btu/SFC)
NO	165-176	163-182
NO_2	87-104	84-97
CO	195-1769	257-1456
Nonstanding pilot, fuel input rate per burner 7950 to 8870 Btu/hr		
	Rich Gas (1022 Btu/SFC)	Lean Gas (983 Btu/SFC)
NO	131-151	135-164
NO_2	75-105	66-76
CO	213-675	213-675
Standing pilot, fuel input rate per burner 9130 to 9890 Btu/hr		
	Rich Gas (1022 Btu/SFC)	Lean Gas(983 Btu/SFC)
NO	163-206	155-177
NO_2	105-117	101-110
CO	180-563	191-670
Gas-fired oven operated at 180°C; Fuel input rate 7970 Btu/hr		
	Average	
NO	56	
NO_2	85	
CO	1898	

The studies compared the bedroom median NO_2 values (2-week averaging time) in Los Angeles and in Boston. Homes with gas stoves showed approximately double the NO_2 concentrations reported for those of homes with electric stoves (40 $\mu g/m^3$ compared to 20 $\mu g/m^3$ in Boston; 80 $\mu g/m^3$ compared to 40 $\mu g/m^3$ in Los Angeles).

In Boston in winter, about 5 percent of the gas-stove homes had bedroom NO_2 averages that exceeded the annual average National Ambient Air Quality Standard (NAAQS) of 100 $\mu g/m^3$. This value fell to less than one percent in summer. In Los Angeles in winter more than 30 percent of homes with gas-stoves exceeded this standard and more than 20 percent exceeded the standard in summer. In both cities less than one percent of the electric-range homes exceeded the standard.

Both studies found that gas stoves, both the pilot light and cooking operation, were a major influence on indoor air quality, adding about 30 to 40 $\mu g/m^3$ of NO_2 on the average. In addition, wall- and floor-vented gas furnaces in the Los Angeles area added roughly equivalent amounts of NO_2.

A study of 152 homes in the Netherlands (Remijn et al., 1985) showed bedroom NO_2 levels ranging between 8 and 66 $\mu g/m^3$, with a geometric mean of 23 $\mu g/m^3$ and a geometric standard deviation of 1.5. (This was reported to be similar to the values observed in Los Angeles and in Boston.) Kitchen and living room levels were higher, with geometric means of 79 and 39 $\mu g/m^3$, respectively, and with a higher geometric standard deviation of 2.2 and 2.0, respectively.

Davidson reviewed the pollutant emission factors for gas stoves presented in the literature. Using these published emission factors, he attempted to determine the important parameters influencing emissions from gas stoves by statistical analysis (Davidson, 1986). His results showed that roughly one-half of the observed variance in the base 10 log of the emission factors for CO could be explained by either poorly adjusted or well adjusted combustion. For NO_2, approximately one-third of the variance could be explained by poor/well adjustments. For NO and NO_x the resulting variance depended on the data subset and ranged from 0.088 to 0.56.

Davidson concluded that burner position and method of sampling were relatively unimportant in explaining variance for CO, NO, NO_2 and NO_x. However, the critical gaps that merit further study were the influence of stove design, gas flow rate, and characteristics of the stove (Davidson, 1986).

Several investigators have indicated that kitchen gas ranges are a major source of indoor air pollution. Such studies, however, address only normal operation of gas ranges for cooking. Sterling and Kobayashi (1981) reports that 50 to 55 percent of New York gas ranges are used for both cooking and heating.

2.2.2.2.2 Gas-fired space heaters. Unvented gas-fired space heaters are reported to contribute to indoor concentrations of NO, NO_2, CO, CO_2, respirable suspended particles (RSP), and HCHO. The factors that influence the emissions include usage pattern, brand of heater, burner design, size of heater (rated input), and the tuning of the fuel to air mixture (U.S. Department of Energy, 1985).

Zawacki et al. (1985) reported emission factors for NO_2, NO, CO, and unburned hydrocarbons from gas-fired space heaters. The factors were experimentally determined for ten different unvented gas space heaters by three different measurement methods (probe, hood, and chamber). The heaters were of various designs (blue-flame with and without radiating tiles, and infrared) and were fueled by either natural gas or liquified propane gas (LPG) and fired over a range of input rates (10,000 to 40,000 Btu/hr).

Zawacki and co-workers initially concluded that emissions were primarily dependent on the heater design (blue-flame versus infrared) and the method of measurement. The probe and hood methods gave almost identical results, which were different from the chamber method, especially for NO and CO. The difference was apparently caused by the reduced oxygen content of the chamber which was held at approximately 0.5 mg/m^3 each (Zawacki et al., 1985). In the final report of this work, Zawacki and co-workers concluded that natural gas-fired infrared heaters averaged NO_2 emissions levels of one-fourth to one-half of that of blue-flame heaters and that these levels appeared to depend on heater design and method of measurement (Zawacki et al., 1986). For blue-flame burners, the average NO_2 emissions levels were essentially the same, about 0.02 $lb/10E^6$ Btu, irrespective of burner input rate and type, fuel type, method of measurement, and existence or absence of suspended radiating tiles (Zawacki et al., 1986).

The propane fired heaters exhibited the highest NO emission levels, which was consistent with expectations that propane heaters developed the highest adiabatic flame temperatures. Natural gas fueled blue-flame burners exhibited about one-half equivalent NO emissions, while natural gas infrared burners produced virtually none. For blue-flame burners, the probe and hood methods of measurement resulted in approximately identical NO emissions (0.1 $lb/10E^6$ Btu) from propane fuel and about one-half as much for natural gas-fired (Zawacki et al., 1986).

Natural gas-fired infrared heaters exhibited the highest average CO emissions levels, 0.03 to 0.17 $lb/10E^6$ Btu, followed by natural gas-fired blue-flame heaters, 0.02 to 0.4 $lb/10E^6$ Btu, and propane-fired blue-flame heaters (0.01 to 0.015 $lb/10E^6$ Btu). The burner design and test method was demonstrated to have a modest to large effect within a category of heaters (Zawacki et al., 1986).

Blue-flame propane heaters exhibited virtually no unburned hydrocarbon (UBHC) emissions, while natural gas blue-flame heaters produced about 0.05 to

0.2 lb/10E^6 Btu, and the infrared tile heaters produced a wide range of UBHC emissions from nearly zero up to 0.9 lb/10E^6 Btu (Zawacki et al., 1986).

The comparison of 8 heaters using the chamber method at Lawrence Berkeley Laboratory (LBL) and the Institute of Gas Technology (IGT) showed no substantial difference in emissions of NO_2, NO and CO (Table 2-5). NO emission were found to be dependent on the quantity of air used for combustion and emissions factors decreased as the chamber humidity increased and as air infiltration decreased. The decreasing air infiltration apparently reduced the chamber oxygen content, inhibiting NO formation and stimulating increased production of CO. The overall effect of water vapor, whether chemical or thermal, is unknown. No effect of either humidity or air on NO_2 emission rates could be detected (Gas Research Institute, 1985).

TABLE 2-5. OVERALL COMPARISON OF EMISSION FACTORS
FROM IGT AND LBL CHAMBER STUDIES

Contaminants	Number of Heaters	Emissions Factors (lb/10E^6 Btu) IGT	LBL
		Blue-Flame Heaters	
NO_2	2	0.0215 ± 0.0025	0.0260 ± 0.0028
NO	2	0.0699 ± 0.0042	0.0663 ± 0.0008
CO	2	0.0260 ± 0.0132	0.0447 ± 0.0194
		Infrared Heaters	
NO_2	5	0.0106 ± 0.0028	0.0119 ± 0.0025
NO	5	0.0005 ± 0.0005	0.0007 ± 0.0004
CO	5	0.1000 ± 0.070	0.0007 ± 0.0004

2.2.2.2.3 Gas-fired water heaters and dryers. Gas water heaters and clothes dryers have not received much attention to date. Although both are designed to be vented, there is the possibility that faulty systems can produce an indoor air impact. The U.S. DOE reports a personal communication with Lawrence Berkeley Laboratory in which it is reported that homes with vented gas water heaters were measured to have indoor concentrations of NO_2 greater than outdoor concentrations. The source in each was traced to flue collars atop the gas water heater (U.S. Department of Energy, 1985).

2.2.2.3 Wood-burning Stoves and Fireplaces. Laboratory and field measurements show that pollutant emission rates from wood combustion in general can vary widely: CO, 4 to 400 g/kg of wood; particles, 0.5 to 63.5 g/kg; total HC, 0.2 to 48.5 g/kg; NO_x, 0.2 to 7.3 g/kg, and particulate organic matter (POM), 0.004 to 0.37 g/kg (Fisk et al., 1985). Consequently, wood-burning stoves, wood-burning furnaces, and fireplaces, even though vented to the outside, can represent a significant source of indoor CO, NO_x, HC, HCHO, and RSP, including carcinogenic POM.

Factors that can affect the indoor emission rates are improper installation, cracks or leaks in stove pipes, negative air pressure within the dwelling, downdrafts, refueling, and accidents. Emissions are also dependent upon the use pattern, manufacturer and type of appliance, and type of wood burned.

There are little data available on the emission rates indoors from such devices. However, they probably emit relatively small quantities of pollutants over long periods of time as a consequence of leaks, and relatively high emissions over short periods such as during reloading. Consequently, the emissions can be expected to vary considerably in terms of mix and frequency.

The Tennesse Valley Authority (TVA) and the Bonneville Power Authority (BPA) undertook to develop data on Indoor Air Quality (IAQ) in relationship to the use of conventional and new technology wood heaters in airtight homes. Four heaters were used in the study: two conventional non-airtight, and two high-technology airtight. Sequential indoor and outdoor measurements of CO, CO_2, NO, and NO_x were taken in a modular test home, evaluated and compared to the National Ambient Air Quality Standards for the same pollutants (Tennessee Valley Authority, 1985).

The non-airtight (NAT) heaters were reported in a TVA/BPA study to represent a significantly larger source of indoor CO_2 than the airtight heaters (AT). The highest hourly indoor CO_2 concentration reported during the test of a NAT wood heater was 1240 ppm, and the highest instantaneous indoor level was 1358 ppm (Tennessee Valley Authority, 1985).

The highest 12-hour indoor SO_2 concentration was 42.4 ppb, which was 30 percent of the 24-hour NAAQS and 8 percent of the 3-hour NAAQS. A statistical analysis of the data showed that the NAT operated with significantly higher indoor SO_2 source strength than the AT unit (Tennessee Valley Authority, 1985).

In a final report, TVA/BPA reported that with respect to NO, the NAT wood heater operated as a much higher indoor NO source strength during closed stack-damper operations when the burn rate was being reduced. Indoor NO_2 levels and source strength were not found to be significantly related to AT or NAT designs. The indoor NO_2 levels were essentially driven by outdoor levels and were generally less than 60 percent of those outdoors (Tennessee Valley Authority, 1986).

Both AT and NAT wood heaters were found to present a statistically significant (at the 0.05 level) source of CO. The source strengths and Indoor/Outdoor Ratio (I/O) for CO were greater for NAT than for AT heaters. The NAT generated much higher indoor CO during closed stack-damper operations. In the final report for this work, TVA/BPA concluded that the maximum 1-hour concentration of CO was 9.1 ppm, and the maximum 8-hour CO concentration was 5.7 ppm (Tennessee Valley Authority, 1986).

In general, the indoor methane hydrocarbons were slightly higher than outdoor levels for both non-burning and active testing, consequently, neither type of wood heater was determined to be a significant source for non-methane hydrocarbons (NMHC) (Tennessee Valley Authority, 1985). However, both were determined to be a source of indoor PAH and B(a)P.

2.2.2.4 Attached Garages. Automobiles or trucks operating in confined small spaces, such as parking garages, can cause extremely high CO concentrations. Wallace reported elevated CO levels in the air and lungs of workers in an underground office that was connected to a parking garage (Wallace, 1983). Even so, there is little in the literature on the affect of automotive exhaust on indoor air quality via an attached garage.

Flachsbart and co-workers employed miniature personal exposure monitors to measure CO in 588 different commercial settings, for example, retail stores, office buildings, hotels, and restaurants in five California cities. They determined that the CO levels indoors were similar to those measured outdoors, usually greater than 0 but less than the NAAQS for 8-hour exposure, unless an indoor source was present. Office buildings with indoor garages were measured to be greater than the NAAQS of 9 ppm (Flachsbart et al., 1984).

Attached garages also present a source of pollutants in addition to the conventional combustion gases. Gammage et al. measured volatile organics inside 40 homes; volatiles being defined as those organics with boiling points of less than 110°C. He determined that the concentration of volatiles was usually ten times higher inside than outside homes. The highly volatile organics were usually dominated by gasoline fumes. Attached garages containing automobiles, stored gasoline, and motor oil were considered to be responsible for this ubiquity. Gammage and co-workers concluded from this work that air drawn through air conditioning ducts located in the attached garages provided an effective means of funneling gasoline and engine exhaust into living spaces (Gammage et al., 1984).

2.2.2.5 Conclusions. A summary table of emission rates for combustion related gases from indoor sources has been published by DOE and is presented in Table 2-6.

TABLE 2-6. POLLUTANT EMISSION RATES (MG/HR)

Source	Appliance Type	NO	NO_2	CO	SO_2
Kerosene Space Heaters	Radiant	0.54-11	16-38	281-542	31-109
	Convective	2-195	33-530	35-635	37-94
Gas Space Heaters		80-4578	3-1225	12-5004	--
Wood Heaters		1.2-3.9	1.3-7.0	70-375	--
Gas Appliances	Range (1 burner)	9.5-455	18-430	191-2700	1.29-1.66
	Oven	30-581	67-270	195-3564	0.67-1.09

Source: U.S. Department of Energy (1985).

The absence of reliable emission factors from the indoor sources makes it impossible to establish a relative ranking between reported sources and their contribution to indoor air pollution at this time. In general, the absence of standardized test procedures, including sampling and analysis methods, and the lack of uniformity in test conditions precludes the use of much of the existing data for the predictive modeling which facilitates risk assessment calculations.

Results from the testing of gas-fired appliances are a good example of the difficulty in making such comparisons. The comparison of various studies of NO, NO_2, NO_x, and CO emissions and concentrations from such sources must be made with great care, since each experiment uses a different sampling point location, appliance, and air-fuel ratio (GEOMET Technologies, Inc., 1976).

Major sources of uncertainty related to indoor air combustion products are the source, identity, and emission rates of unburned benzene hydrocarbons (UBHC); the reactivity of the various organic and inorganic gases; and the applicability of a given control option to a specific pollutant source.

2.2.2.6 Major Knowns and Unknowns. The most important research need is accurate source characterization, including measurements of emissions from combustion sources, pollutant removal by indoor "sinks," and the factors that affect their emission or removal rates. The ultimate objective of source characterization is to determine the most important sources of indoor air pollutants, and to characterize them in such a way that the most cost-effective methods of controlling them can be determined.

Indoor air source characterization research needs related to combustion sources include the following:

- Expand the characterization of organics from combustion devices,
- Survey indoor combustion sources to gather statistics on age, condition, and operational parameters,
- Investigate the factors affecting leakage rates of pollutants from vented combustion sources,
- Study sink rates of pollutants, especially NO_2 and SO_2, on indoor materials, and
- Measure source strengths of automobile exhausts in attached garages.

2.2.3 Carbon Monoxide

Carbon monoxide is a colorless, odorless, tasteless gas that is slightly soluble in water. It is slightly lighter than air (specific gravity = 0.967). It is an asphyxiant for which the mechanism of action is an avid binding to hemoglobin. Its affinity for hemoglobin is about 210 to 250 times that of oxygen, thereby interfering with oxygen transport to tissues and resulting in tissue hypoxia.

2.2.3.1 Exposure. Human exposures to CO in the residential indoor microenvironment have been investigated in studies in which subjects have carried personal monitors that recorded an average CO exposure during intervals of up to one hour while a subject is indoors at home, or at work in a public building

(Akland et al., 1985). The results are reported in several publications and EPA reports (Holland, 1983; Akland et al., 1986). Table 2-7 shows the mean and standard deviations of CO in the several indoor microenvironments defined in the study. These highly skewed results indicate that people may be exposed to CO values that approach the NAAQS for both the 8-hour and 1-hour averaging times. However, these data were collected during the winter and do not represent an annual average for these cities. Table 2-8 gives the CO distribution found in the indoor residential microenvironment. The 8-hour NAAQS concentration for CO of 9 ppm was exceeded in 4.3 percent of the homes.

TABLE 2-7. INDOOR MICROENVIRONMENTS LISTED IN DESCENDING ORDER OF WEIGHTED MEAN CO CONCENTRATION

Indoor Microenvironment Category Measured	Number of subjects	CO Concentration (ppm) Mean	Std. Dev.
Public garage	116	13.46	18.14
Service station or motor vehicle repair facility	125	9.17	9.33
Other location	427	7.40	17.97
Other repair shop	55	5.64	7.67
Shopping mall	58	4.90	6.50
Residential garage	66	4.35	7.06
Restaurant	524	3.71	4.35
Office	2287	3.59	4.18
Auditorium, sports arena, concert hall, etc.	100	3.37	4.76
Store	734	3.23	5.56
Health care facility	351	2.22	4.25
Other public buildings	115	2.15	3.26
Manufacturing facility	42	2.04	2.55
Residence	21543	2.04	4.06
School	426	1.64	2.76
Church	179	1.56	3.35

2.2.3.2 Monitoring of CO. From a health effects assessment perspective, there is a need to measure CO levels in the air which people breathe on a continuous, or real-time, basis. The most common method for continuous CO monitoring in ambient air is based on nondispersive infrared (NDIR) spectroscopic detection. While sensitive enough for the purposes, NDIR instruments are much too bulky and complicated for personal exposure monitoring. To address this need, therefore, a variety of small, portable personal monitors were developed during

TABLE 2-8. INDOOR RESIDENTIAL CO DATA FOR
DENVER, COLORADO - WINTER 1982-83
21,229 OBSERVATIONS UNWEIGHTED

Percentile	Concentration (ppm)
10%	0.5
20	0.8
30	1.0
40	1.3
50	1.8
60	2.3
70	3.0
80	4.2
90	6.2
95	8.5
99	14.5
Maximum	26.7
Mean	2.76
Standard Deviation	2.92

the past decade (Ott et al., 1986b). Most of these CO personal exposure monitors (PEMs) were developed for occupational safety and health uses, especially by the mining industry. Most of the devices are based on electrochemical detection; that is, they employ a liquid or solid electrolyte in which CO is converted to CO_2, thereby generating an electrical signal. The signal is proportional to the quantity of CO present in the gas stream, and the continuous electrical signal is either recorded internally or displayed on a digital readout system. A small pump operates continuously to send air into the sensing cell, and chemical filters in the intake stream remove interference.

The most promising of the electrochemical CO PEMs was one developed by General Electric Company and called "COED" (Ott et al., 1986a). In the winter of 1982-83, the EPA successfully demonstrated the utility of two versions of this new monitor in large-scale pilot studies of statistically representative samples of the populations in two cities, Denver, CO, and Washington, DC (Akland et al., 1985). The CO PEM used in Denver contained a sensing cell with a solid polymer electrolyte, and an internal microprocessor data logger which recorded and stored up to 100 readings from the PEM (Ott et al., 1986b). This version of the CO PEM, designated the COED-I, weighed less than 4.4 lg (2 lb.) and included a digital readout display. The second version of the CO PEM utilized in the Washington study (Mack et al., 1987) incorporated the same CO detection system, but used an external data logging system built around a user-programmable pocket calculator. This prototype system, designated the COED-II, was much more flexible and stored more than 400 data entries. The COED-II was capable of operating continuously for 40 hours and provided accurate measurements at 2 ppm sensitivity. During the Washington study, more than 1200 exposure profiles were constructed using the COED-II, and, considering the newness and complexity of the technique, the problems encountered were relatively minor.

While the COED-II monitor functioned well, it required daily servicing. A self-controlled data-logging PEM that can be operated unattended for two weeks or more is needed for further Total Human Exposure (THE) and IAQ surveys. For large surveys, use of the COED-II or a similar active (pumped) PEM would be cost prohibitive. Therefore, a sensitive and reliable passive device needs to be developed.

2.2.3.3 Health Effects. The effects of CO on oxygen transport are well established (National Research Council, 1977; U.S. Environmental Protection Agency, 1979, 1984a). CO interferes with oxygen transport by avidly binding to hemoglobin to form carboxyhemoglobin (COHb) and by shifting the oxyhemoglobin dissociation curve to the left. As a result of this shift, oxygen that is attached to hemoglobin is released to the tissues less readily, and additional tissue hypoxia results. The health effects of low levels of CO exposure (COHb <3 percent) are controversial, but the problem of CO poisoning by indoor combustion sources has been well described. The clinical manifestations of poisoning by CO primarily represent the effects of reduced oxygen transport to tissue and organs with high oxygen demand, such as the heart and the brain. The neurological manifestations range from impaired mentation and behavioral alterations to coma (Dolan, 1985; Ginsberg, 1985). Cardiac effects include arrhythmias and myocardial infarction (Dolan, 1985). Even low levels of carbon monoxide exposure affect the heart and brain.

Research on the health effects of lower levels of carbon monoxide has primarily been conducted with experimental exposures of subjects, rather than with epidemiological methods. Studies of carbon monoxide exposures in indoor environments have been reported (Wallace, 1983; Cox et al., 1985), and health effects of indoor exposure to carbon monoxide at levels lower than those causing poisoning have occasionally been reported.

Tissue hypoxia resulting from the reduced oxygen carrying capacity of the blood is generally thought to be the main mechanism of action underlying the induction of toxic effects of low-level CO exposures (5 to 20 percent COHb). However, the precise mechanisms by which toxic effects are induced via COHb formation are not yet fully understood. Alternative mechanisms of CO-induced toxicity (besides COHb) have been hypothesized (Coburn, 1979; Agostoni et al., 1980), but none has yet been demonstrated to operate at relatively low CO exposure levels. Blood COHb levels, then, are currently accepted as representing a useful physiological marker by which to estimate internal CO burdens due to the combined contribution of (1) endogenously derived CO and (2) exogenously derived CO resulting from exposure to external sources of CO. COHb levels likely to result from particular patterns (concentrations, durations, etc.) of external CO exposure can be reasonably well estimated from equations developed by Coburn et al. (1965).

Evaluation of human CO exposure situations indicates that occupational exposures in some workplace situations can regularly exceed 100 ppm CO, often leading to COHb levels of 10 percent or more. In contrast, such high exposure levels are much less commonly encountered by the nonoccupationally exposed general public. More frequently, exposures to less than 25 ppm CO for extended periods of time occur among the general population and, at the low exercise levels usually engaged in under such circumstances, resulting COHb levels most typically remain 2 to 3 percent among nonsmokers. Those levels can be compared to the physiologic norm for nonsmokers, which is estimated to be in the range of 0.3 to 0.7 percent COHb. Baseline COHb concentrations in smokers, however, often greatly exceed 3 percent, reflecting absorption of CO from inhaled smoke.

Some health effects associated with CO exposure occur at rather low COHb levels (See Table 2-9). Four types of health effects reported or hypothesized to be associated with CO exposures (especially those producing COHb levels below 10 percent) were evaluated in the addendum to the 1979 Air Quality Criteria Document for Carbon Monoxide (U.S. Environmental Protection Agency, 1984a): (1) cardiovascular effects; (2) neurobehavioral effects; (3) fibrinolysis effects; and (4) perinatal effects. Available data demonstrate an association between cardiovascular and neurobehavioral effects at relatively low-level CO exposures. Evidence that is less clear suggests that other types of health effects are associated with low-level CO exposures. In regard to cardiovascular effects, decreased oxygen uptake capacity and resultant decreased work capacity under maximal exercise conditions have been clearly shown to occur in healthy young adults starting at 5.0 percent COHb; and several studies

TABLE 2-9. LOWEST OBSERVED EFFECT LEVELS FOR HUMAN HEALTH EFFECTS ASSOCIATED WITH LOW LEVEL CARBON MONOXIDE EXPOSURE

Effects	COHb concentration (Percent)[a]	Reference
Statistically significant decreased (~3 to 7 percent) work time to exhaustion in exercising young healthy men	2.3 to 4.3	Horvath et al. (1975) Drinkwater et al. (1974)
Statistically significant decreased exercise capacity (i.e., shortened duration of exercise before onset of pain) in patients with angina pectoris and increased duration of angina attacks	2.9 to 4.5	Anderson et al. (1973)
Statistically significant decreased maximal oxygen consumption and exercise time during strenuous exercise in young healthy men	5 to 5.5	Klein et al. (1980) Stewart et al. (1978) Weiser et al. (1978)
No statistically significant vigilance decrements after exposure to CO	Less than 5	Haider et al. (1976) Winneke (1973) Christensen et al. (1977) Benignus et al. (1977) Putz et al. (1976)
Statistically significant diminution of visual perception, manual dexterity ability to learn, or performance in complex sensorimotor tasks (such as driving)	5 to 17	Bender et al. (1971) Schulte (1973) O'Donnell et al. (1971) McFarland et al. (1944) McFarland (1973) Putz et al. (1976) Salvatore (1974) Wright et al. (1973) Rockwell and Weir (1975) Rummo and Sarlanis (1974) Putz et al. (1979) Putz (1979)
Statistically significant decreased maximal oxygen consumption during strenuous exercise in young healthy men	7 to 20	Ekblom and Huot (1972) Pirnay et al. (1971)

[a] The physiologic norm (i.e., COHb levels resulting from the normal catabolism of hemoglobin and other heme-containing materials) has been estimated to be in the range of 0.3 to 0.7 percent (Coburn et al., 1963).

Source: U.S. Environmental Protection Agency (1984a).

revealed small decreases in work capacity at COHb levels as low as 2.3 to 4.3 percent. These cardiovascular effects may have health implications for the general population in terms of potential curtailment of certain physically demanding occupational or recreational activities under circumstances of sufficiently high CO exposure. However, of greater concern at more typical ambient CO exposure levels are certain cardiovascular effects (i.e., aggravation of angina symptoms during exercise) likely to occur in a smaller, but sizeable, segment of the general population. This group, chronic angina patients, is presently viewed as the most sensitive risk group for CO exposure effects, based on evidence for aggravation of angina occurring in patients at COHb levels of 2.9 to 4.5 percent COHb. Such aggravation of angina is thought to represent an adverse health effect for several reasons articulated in the 1980 proposal preamble (Federal Register, 1980); the Clean Air Scientific Advisory Committee (CASAC) concurred with EPA's judgment on this matter. Dose-response relationships for cardiovascular effects in coronary artery disease patients remain to be more conclusively defined, and the possibility cannot be ruled out at this time that such effects may occur at levels less than 2.9 percent COHb (as hinted at by the results of the now questioned Aronow studies). New studies are, therefore, currently in progress to investigate the effects of CO on aggravation of angina at levels in the range of 2 to 4 percent COHb.

No reliable evidence demonstrating decrements in neurobehavioral function in healthy young adults has been reported at COHb levels less than 5 percent. Much of the research at 5 percent COHb did not show any effect even when behaviors similar to those affected in other studies were involved. However, if any CO effects on neurobehavioral functions in fact occur less than 5 percent COHb, then none of the significant-effects studies would have found such decrements, because none of them used COHb levels less than 5 percent. Other workers who failed to find CO decrements at 5 percent or higher COHb levels may have employed tests not sufficiently sensitive to reliably detect small effects of CO. From the empirical evidence, then, it can be said that the COHb levels in the 5 percent range and greater produce decrements in neurobehavioral function. However, it cannot be said confidently that COHb levels less than than 5 percent would be without effect. One important point made in the 1979 document should be reiterated here. Only young, healthy adults have been studied using demonstrably sensitive tests and COHb levels at 5 percent or greater. The question of groups at special risk for central nervous system (CNS) effects, therefore, has not been explored. Of special note are those individuals who are taking drugs which have primary or secondary depressant effects which would be expected to exacerbate CO-related neurobehavioral decrements. Other groups at possibly increased risk for CO-induced neurobehavioral effects are the elderly and ill, but these groups also have not been evaluated for such risk.

Only relatively weak evidence points toward possible CO effects on fibrinolytic activity and, then, generally only at rather high CO exposure levels. Similarly, whereas certain data also suggest that perinatal effects (e.g., reduced birth weight, slowed postnatal development, sudden infant death syndrome) are associated with CO exposure, only insufficient evidence exists by which to either confirm such association qualitatively or to establish any pertinent exposure-effect relationships.

Based on currently available scientific evidence, onset of adverse health effects occur, even in healthy individuals, at COHb levels in the range of 5 to 20 percent and greater. There are, however, uncertainties regarding the extent to which adverse health effects are occurring in a large number of sensitive individuals with COHb levels in the range of 3 to 5 percent. These uncertainties were a major reason for EPA's decision not to change the existing 8-hour primary standard level of 9 ppm (Federal Register, 1985). Additional studies are now under way to identify the relationship between COHb and aggravation of preexisting cardiovascular disease. The new information obtained from these studies will allow a better determination of the level of COHb necessary to cause adverse effects in the sensitive population and ultimately set an appropriate level for the CO NAAQS.

Angina patients or others with obstructed coronary arteries, but not yet manifesting overt symptomatology of coronary artery disease, appear to be best established as a sensitive group within the general population that is at increased risk for experiencing health effects (i.e., exacerbation of cardiovascular symptoms) of concern at ambient or near-ambient CO exposure levels. Several other probable risk groups were identified, including: (1) fetuses and young infants; (2) the elderly (especially those with compromised cardiopulmonary functions); (3) younger individuals with severe cardiac or acutely severe respiratory diseases; (4) individuals with preexisting diseases such as chronic bronchitis, emphysema, or congestive heart failure; (5) individuals with hematological diseases (e.g., anemia) that affect oxygen-carrying capacity or transport in the blood; (6) individuals with genetically unusual forms of hemoglobin associated with reduced oxygen-carrying capacity; and (7) individuals using medicinal or illicit drugs having CNS depressant properties. However, there is currently little empirical evidence available by which to specify health effects associated with ambient or near-ambient CO exposures. Nor does unambiguous evidence exist which clearly establishes that healthy nonsensitive individuals. Those in the above probable risk categories are affected at lower CO exposure levels under high altitude conditions than CO exposure concentrations effective at lower altitudes.

2.2.3.4 Conclusions.

Major Knowns

1. CO causes cardiovascular effects. Chronic angina patients are the most sensitive risk group currently known. Effects have been observed at 2.9 to 4.5 percent COHb.
2. Higher levels of CO resulting in ≥ 5 percent COHb can cause decrements in neurobehavioral functioning.
3. Several other types of health effects are observed after CO exposure, but they are not as well defined as the cardiovascular effects.

Major Unknowns

1. Patients with ischemic heart disease (Coronary Artery Disease - CAD) who are exposed to low levels of CO have been reported to experience persistent angina (chest pain) at COHb levels of approximately 2 percent. Studies need to be completed to verify this report and to determine if all CAD patients or sensitive segments of this population are at increased risk to CO.
2. While the incidence of angina pectoris has been identified as a potential major health risk in patients with ischemic heart disease, little is known about the reproducibility or recurrence of this phenomena. Such information is vital to a proper interpretation of the health data on CO effects.
3. A second group that may be at increased risk to low levels of CO exposure and which needs to be studied are those with congestive heart failure.
4. Patients with ischemic heart disease need to be studied to determine whether they will develop cardiac arrhythmias following exposure to CO.

2.2.4 Nitrogen Dioxide

Nitrogen dioxide, which is a red to brown gas with a pungent, acrid odor, is produced during combustion at high temperatures from the combination of nitrogen and oxygen from air. It is an oxidizing agent that is highly irritating to mucous membranes, and causes a wide variety of health effects.

2.2.4.1 Exposure. Human exposures to NO_2 in indoor microenvironments have been obtained primarily from indoor concentration studies and PEM studies in which the exposure is integrated over both indoor and outdoor microenvironments. Thus it is difficult to construct a data set of NO_2 observations alone obtained while the subject is in the target microenvironment which fits the criterion of exposure to a single pure compound. All studies show that indoor NO_2 levels are lower than the immediate outdoor values when there are no indoor sources (e.g., all-electric homes) due to the reactivity of NO_2 with indoor surface. In the presence of an indoor source such as a cigarette or a gas stove in use, the NO_2 levels are invariably higher than the outdoor NO_2. However, for the cases of intermittent usage of the source, the all-electric values approach the gas home values when the combustion sources are used infrequently. Figure 2-1 presents mean indoor microenvironment NO_2 values from a published study that measured indoor and outdoor concentrations simultaneously (Spengler et al., 1979). Short-term average NO_2 concentrations can reach ~1000 $\mu g/m^3$ in gas homes during cooking in the kitchen.

2.2.4.2 Monitoring. Two main passive devices are available for measuring integrated exposure to NO_2: the Palmes tube and the Yanagisawa badge. The Palmes tube (Palmes et al., 1976) is an acrylic or metal tube, normally about 1 cm in diameter and 7 cm long, containing three stainless steel grids coated with triethanolamine (TEA) in a cap at the top of the tube. The bottom of the tube is open to sample the air, allowing NO_2 to diffuse upward until it reacts with the TEA, forming a stable complex for subsequent analysis by spectrophotometry. Although the sample has a sensitivity of several hundred ppb-hours, with a lower limit of detection of 300-ppb-hr, requiring a minimum 2-day collection time at normal indoor environmental levels, most studies use 1- to 2-week collection times. The Palmes tube has been used to study the relationship between respiratory illness in children and the use of gas stoves for cooking (Goldstein et al., 1979).

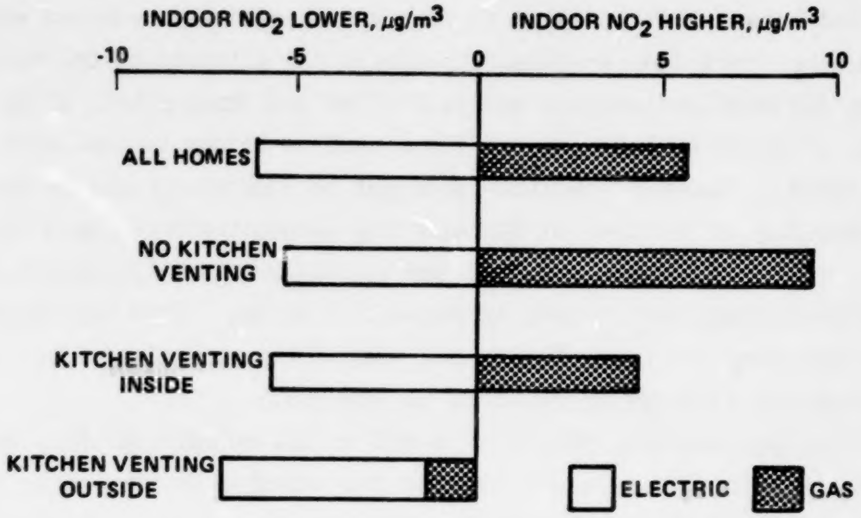


Figure 2-1. Mean indoor/outdoor difference in nitrogen dioxide concentrations from cooking fuel and kitchen ventilation, average across all indoor/outdoor sites (May 1977-April 1978).

Source: Spengler et al. (1979).

In an attempt to lower the minimum detection limit of the Palmes tube sufficiently to achieve 8- to 24-hr utility, an ion chromatographic (IC) analytical finish was explored. The application of IC, which is inherently more sensitive than UV spectrophotometry, combined with a specially-designed concentrator column, improved the overall sensitivity by an order of magnitude to 30 ppbv-hr + 20 percent (Miller, 1987, in press). While this analytical improvement would theoretically permit one day or shorter exposure times, the concentration step caused chloride ion interference with resolution of the nitrite ion. It became necessary to build a special extraction apparatus to extract the nitrite ion from the triethanolamine-coated screens in order to prevent handling contamination. Finally, the acrylic tube of the Palmes NO_2

device was found to act as a sink or a source of NO_2 depending on how well it was cleaned. Stainless-steel tubes were investigated to minimize contamination and improve the minimum detection limit, but these proved too cumbersome to recommend for routine personal monitoring.

A second method that appeared to have the sensitivity required was based on a Thermosorb cartridge originally designed for nitrosamine collection and analysis by GC-chemiluminescence analysis (Fine and Rounbehler, 1975). The feasibility of converting the pumped Thermosorb cartridge to the passive mode was investigated. However, results could not be replicated due to problems with the chemistry of reaction of NO_2 with the morpholine-based sorbent.

A recent third approach modified the stainless-steel PSD developed for volatile organic chemicals to make it responsive to NO_2. This was accomplished simply by replacing the Tenax sorbent bed with TEA-coated glass fiber filters. Ion chromatography (IC) can be employed for analysis.

The effective sampling rate of this PSD is 154 cm^3/min for NO_2, making it potentially 150 times more sensitive than the standard Palmes tube. These devices have been evaluated in an NO_2 exposure chamber at concentrations ranging from 20 mg/m^3 to 460 mg/m^3 (10 to 250 ppbv) and compared with a continuously-reading Bendix NO_x chemiluminescent monitor. For 24-hr exposures, the correlation coefficient was 0.9955 over the range studied. Nitric oxide (91 mg/m^3)caused no deleterious effect on the efficiency of the PSD at 57 percent RH. The response was also shown to be linear over exposure times from 2 to 30 hours at constant air concentrations (109 mg/m^3 or 58 ppbv). The minimum detection limit for the PSD was shown to be 30 ppb-hr when IC analysis was performed without the aid of a concentrator column. The PSD is a very small, dual-faced cylinder, measuring 3.8 cm (1.5 in) o.d. x 1.2 cm (7/16 in) and weighing only 36 g (1.3 oz). The device is undergoing further testing to determine potential interferences from other chemical species. Parallel comparisons with the Luminox LMA-3 monitor also need to be conducted.

The Yanagisawa badge (Nitta and Maeda, 1982) uses an absorbent sheet of cellulose fiber coated with TEA. A five-layer mat of hydrophobic fiber reduces the effect of face velocity on the diffusion characteristics of the badge. The sensitivity of the badge is 66 ppb-hours, about ten times more sensitive than that of the Palmes tube. Thus, sampling periods as short as eight hours at environmental levels are possible. The badge has been used in a study of the effect of unvented heaters on indoor NO_2 levels (Yanagisawa et al., 1981).

As in the case of CO, there is a considerable need for monitoring systems capable of determining continuous exposures to NO_2 in air. Systems designed for outdoor ambient air monitoring are too large and complex to be used for THE or IAQ monitoring purposes. While continuous monitoring is required to determine brief exposures to NO_2 encountered during cooking with gas appliances, more sensitive methods to measure low level, time-weighted average concentrations are also needed for complete assessment of potential health effects.

Over the past three years, considerable progress has been made in the development of portable real-time NO_2 monitors based on the chemiluminescent (light-producing) reaction between NO_2 and luminol (Wendel et al., 1983). Air entering the system is pulled through a unit and across the face of a filter wetted with a solution containing luminol. The chemiluminescence is detected on a photodiode, which produces a voltage signal proportional to the NO_2 concentration. Recent research and development sponsored by EPA has led to the commercialization of a small, light-weight monitor known as the Luminox LMA-3 (Schiff et al., 1986). The monitor was tested by EPA and found to have a significant temperature dependence. Subsequent redesign has been successful in moderating this dependence and existing units are currently being retrofitted at the manufacturer's expense. Ultimate acceptance of the unit as a useful means for directly monitoring indoor NO_2 is expected if remaining questions concerning the characteristics of the luminol reactant solution can be treated. The exact composition of the solution determines the response characteristics. During FY 87 the solution will be standardized to comply with the sensitivity and linearity requirements of indoor air monitoring.

The Luminox LMA-3 monitor is 38 x 20 x 22 cm (15 x 8 x 8.5 in.) in size and weighs 7 kg (15.5 lb), which makes it easily transportable for an adult. However, it needs to be operated on 115 VAC power, as the internal batteries will only permit three hours of operation. It appears unlikely that the luminol-based monitoring technology will permit significant improvements in portability or battery operation. Consequently, further attempts at miniaturization of the Luminox monitor have been abandoned in favor of pursuing electrochemical sensor development.

A prototype electrochemical sensor system was recently developed by Transducer Research, Inc. (TRI). With EPA support, TRI has significantly enhanced sensitivity and demonstrated a total signal drift of less than 25 ppbv over 24 hours for the device. Interference from sulfur compounds remains a problem and will be addressed as part of the development cycle. The FY 87 objectives include fabrication of units suitably packaged for testing and evaluation as PEMs.

Improved PSDs for NO_2 have been developed in a parallel effort with continuous NO_2 monitor development. PSDs present no portability problems and, if sufficiently sensitive, could serve as complementary tools for overall assessment of NO_2 exposures.

Two other active NO_2 monitors are currently being tested for indoor sampling application. The first is a small detector made by Interscan which uses an electrochemical cell and is designed for use in occupational settings. It has been modified by Harvard University and is being tested as a PEM to measure concentrations found in ambient settings. A miniature data system is also under test to permit assessment of the exposure distributions. The Ecolizer, made by Ecological Sciences, Inc., is currently being tested at Columbia University to study gas stove emission exposures. It is small enough to be worn on the person.

2.2.4.2.1 Absorption of NO_2. Several studies have shown that various materials including fabrics, paints, metals, and tatami mats absorb NO_2. The extent of absorption by a typical home can be estimated by comparing outdoor NO_2 values with those in homes with no sources. Future studies needed in this area include personal monitoring studies to answer the difficult question of the influence of indoor concentrations on total exposure.

2.2.4.3 Health Effects of NO_2. The health effects of NO_2 have been studied extensively and reviewed by the U.S. Environmental Protection Agency (1982b) and the World Health Organization (1987). Because of adverse health effects associated with NO_2, EPA has established a NAAQS of 0.05 ppm as an annual average.

Knowledge of the health effects of NO_2 are derived from animal toxicological, human clinical, and epidemiological studies. The animal and human clinical data will be summarized briefly here. The discussion of the epidemiology studies will be expanded because they are essentially studies of the effects of the use of gas stoves and therefore the information is more broadly useful to indoor air, since it is source-specific information.

Nitrogen dioxide, as described earlier in this chapter, is produced by many combustion sources within homes as part of a very complex mixture from a given combustion source. However, with rare exception, controlled studies of NO_2 (i.e., very controlled laboratory studies in which an animal or man is exposed to known levels of NO_2 and effects are measured) are with NO_2 alone. Thus, we have no significant knowledge of additivity, synergism, or antagonism associated with NO_2 in mixtures with other combustion products, much less with the full range of indoor air chemicals. The only exceptions with controlled studies are with other common chemicals, namely O_3, NO_2, and SO_2 (U.S. Environmental Protection Agency, 1982b; Mustafa et al., 1984). The few studies of the interaction of NO_2 and O_3 indicate that NO_2 has either no influence, is additive, or is synergistic, depending upon the exposure regimen and end point chosen. Human clinical studies of NO_2 in association with O_3 and/or SO_2 show no contribution of NO_2 to the effects, under the protocol used. Several mixture studies with NO_2 were performed with animals in the 1960s and early 1970s, but the study designs do not permit an interpretation of the role of NO_2. The controlled interaction studies clearly indicate that when we have knowledge of the health risks of NO_2, this knowledge cannot be used to quantitatively estimate the risks of NO_2 in mixture with other pollutants, including indoor pollutants. It can only be roughly assumed that the effects would be a combination of additivity and synergism. With this as background, a summary of the effects of NO_2 in controlled human and animal studies follows.

Hundreds of animal toxicology studies have been performed with NO_2 (U.S. Environmental Protection Agency, 1982b; World Health Organization, 1987). Clearly, high levels of NO_2 (>2.5 ppm) cause increased susceptibility to pulmonary bacterial infections and other changes in host defenses (acute exposure), major structural alterations in the lung, including emphysemic-like effects in several animal species (subchronic and chronic exposure), changes in pulmonary function (subchronic exposure), and changes in lung biochemistry (acute, subchronic, and chronic exposure). Lower levels of NO_2 (<1 ppm) also cause decrements in lung host defenses (subchronic and chronic exposure), lung structural changes that can be interpreted as possibly indicative of the development of chronic lung disease (subchronic exposure), and changes in lung metabolism (acute exposure). Very young animals, compared to young adults, do not appear to be more susceptible to the subchronic effects of NO_2. Extrapulmonary effects have also been observed at <1 ppm NO_2 (acute, subchronic, and chronic exposure). Generally, the animal toxicology studies form the main basis for the concern that chronic exposure of man to NO_2 could well result in chronic lung disease, likely to be irreversible, and that the concentration of NO_2 is more of a determinant of health effects than is the length of exposure, although length of exposure also plays a significant role.

Human clinical studies have demonstrated that acute (<2 hr) exposure of resting, normal subjects only causes changes in pulmonary function at levels rarely, if at all encountered indoors (>2 ppm) (U.S. Environmental Protection Agency, 1982b). More recent studies are summarized in Table 2-10. A single more recent study (Bylin et al., 1985) has reported pulmonary function changes of normal subjects exercising in 0.2 ppm NO_2, but this is in contrast to recent reports by others (Linn et al., 1985). Current research has identified a susceptible subpopulation, that is, asthmatics (Table 2-10). Two institutions have observed decrements in pulmonary function in asthmatics exercising in an atmosphere of 0.3 ppm NO_2 (Horstman et al. 1987; Bauer et al., 1986); another study did not find asthmatics to be more sensitive (Linn et al., 1985). These and other studies also found that NO_2-exposed asthmatics were also more sensitive to bronchoconstrictor agents administered in the laboratory. The emerging information currently indicates that asthmatics are a sensitive subpopulation, but there is a variability in the sensitivity of asthmatics, with an unknown causative factor for this variability.

As mentioned earlier, the literature on the epidemiology of NO_2 is based on the study of people exposed to gas stoves that emit NO_2. Gas stoves emit other pollutants as well, but many experts interpret the effects to be due to NO_2. For the purposes of assessing risks of exposures to indoor air, however, the causative factor of effects in these studies is more directly and clearly interpretable in terms of the source, independent of any single chemical emitted from that source. It should be noted that of all the combustion appliances, gas stoves are the only ones that have been well-studied.

In most of the epidemiological investigations, school-age children have been the subjects, and symptom status and retrospective illness histories have been obtained by parent-completed questionnaires. In some studies, a measure of lung function has been obtained. In these investigations, the source of NO_2 exposure has primarily been emissions from gas-fueled cooking stoves. Exposure has most often been categorized by simple questions concerning type of cooking fuel; NO_2 levels have been directly measured in only a few of the investigations. Stove type alone does not predict average exposures nor does it provide any information concerning peak exposures during stove operation.

TABLE 2-10. CONTROLLED HUMAN EXPOSURE STUDIES OF NO_2 EFFECTS

Pollutant/ Concentration	Duration of Exposure and Activity	Number of Subjects and Type	Pulmonary Effects	Symptoms	References
NO_2 0.1 ppm	1 hr at rest	20 asthmatics 20 normals	SGaw decrease induced by carbachol significantly enhanced in normals ($p < 0.005$) and also in asthmatics ($p < 0.05$).	None	Ahmed et al. (1982)
NO_2 0.1 ppm	1 hr at rest	9 asthmatics hypersensitive to ragweed	NO_2 had no significant effect on baseline SGaw, FEV_1, and V_{isov}.	None	Ahmed et al. (1983)
NO_2 0.1 ppm	1 hr at rest	15 normals 15 asthmatics (atopics)	No statistically significant change in airway resistance (SRaw) for either group.	None	Hazucha et al. (1983)
NO_2 0.15 ppm O_3 0.15 ppm $(NO_2 + O_3)$ 0.15 ppm	2 hr intermittent light exercise	6 normals	Significant decrease (>5%) in Gaw/Vtg with O_3 for 5 of 6 subjects and all six for combined O_3-NO_2; very small (<5%) decreases in Gaw/Vtg. with NO_2 alone in 3 of 6 subjects.	Cough with O_3 and $O_3 + NO_2$ but not NO_2 alone.	Kagawa and Tsuru (1979)
NO_2 0.2 ppm	2 hr intermittent light exercise	31 asthmatics	No effect on forced expiratory function or total respiratory resistance observed with NO_2 alone. Statistically significant small ($X \cong 5.5\%$) exacerbation by NO_2 of metacholine-induced bronchoconstriction in 17 of 21 subjects tested.	Significantly fewer symptoms during NO_2 exposure compared to air ($p < 0.05$).	Kleinman et al. (1983)
NO_2 4.0 ppm	75 min with two intermittent exercises (V_E = 25 l/min and 50 l/min)	23 asthmatics, 25 normals	Small increases in SRaw following exercise in both clean air and NO_2. No significant difference between the two exposure conditions.	None	Linn et al. (1985)

(continued on following page)

TABLE 2-10. (continued)

Pollutant/ Concentration	Duration of Exposure and Activity	Number of Subjects and Type	Pulmonary Effects	Symptoms	References
NO_2 0.3 ppm	30 min with one 10 exercises ($\dot{V}_E$ = 30 l/min)	15 asthmatics	Greater than 75% lung deposition during both rest and exercise. No responses to at rest exposures. decreases in FEV_1 and PEFR at 60% TLC were significantly greater following exercise in NO_2 than that in clean air. Small, but significant increase in airway reactivity (bronchoconstrictor response) to cold air provocation.	None reported	Bauer et al. (1984)
NO_2 0.3 ppm	90 min with three intermittent exercises ($\dot{V}_E$ = 45 l/min)	13 asthmatics	Decrease in FEV_1 following initial 10 min exercise in NO_2 was significantly longer than that observed in clean air. After the second and third exercise, SRaw in- and FEV_1 and FVL decreases significantly greater in NO_2 than in clear air.	Slight cough and dry mouth/ throat after initial exercise in NO_2	Rogers et al. (in press)
NO_2 0.12 ppm NO_2 0.24 ppm NO_2 0.5 ppm	20 min at rest	8 normals 8 asthmatics	In normals, increased SRaw at 0.24 ppm and decreased SRaw at 0.5 ppm; no effect on TGV. In asthmatics, no significant effect on SRaw; lower TGV after 0.5 ppm. In both groups, no significant change in respiratory rate. Bronchial reactivity to histamine studied after 0.5 ppm: increased reactivity (SRaw) in asthmatics, but not normals.	Not studied	Bylin et al. (1985)

SRaw = Specific Airway Resistance

SGaw = Specific Airway Conductance

Source: Grant (1984).

Consistent evidence of excess respiratory symptoms and illnesses in children living in homes with gas stoves has not been found (Table 2-11). Melia and co-workers (1977) published one of the first reports of adverse health effects associated with exposure to gas stove emissions. Significantly higher prevalences of bronchitis, day or night cough, and colds going to the chest were found for English schoolchildren living in homes with gas as compared with electric stoves. In follow-up assessments of these children, the pattern of an adverse effect of gas stove exposure was less evident (Melia et al., 1979). Other studies in England by this and other groups have not shown consistent evidence of harmful effects of indoor NO_2 exposure on symptoms and illnesses in children (Florey et al., 1979; Melia et al., 1982a,b, 1983; Ogston et al., 1985).

In a 1980 report from Harvard's Air Pollution Health Study in six U.S. communities, the initial findings of the British investigators were corroborated (Speizer et al., 1980). In a cross-sectional study of 8,120 children ages 6 to 10 years, report of a serious respiratory illness before age two years was weakly but significantly (odds ratio = 1.23) associated with current use of a gas stove. However, with expansion of the cohort to 10,106 children, the odds ratio for respiratory illness before age two declined to 1.13 ($p = 0.07$) (Ware et al., 1984).

Other investigations, both cross-sectional and longitudinal, have also examined the associations between gas stove exposure and respiratory illnesses and symptoms in children. The findings do not consistently provide evidence of effects of gas stove exposure (Keller, 1979a,b; Dodge, 1982; Ekwo et al., 1983; Schenker et al., 1983).

The data concerned with lung function level in children are similarly inconclusive. Of four investigations with large sample sizes (Ware et al., 1984; Speizer et al., 1980; Hasselblad et al., 1981; Vedal et al., 1984) two have demonstrated unequivocal and statistically significant effects (Speizer et al., 1980; Hasselblad et al., 1981). The magnitude of effect was extremely small, on average. While lung function has been considered in other studies, the sample sizes were inadequate for detecting effects of the magnitude found in the larger studies (Table 2-12).

Only a few investigations provide data on effects of indoor NO_2 on adults. Prospective studies of acute respiratory illness occurrence have not demonstrated excesses in residents of homes with gas stoves (Keller et al., 1979a,b; Love et al., 1982). Studies of lung function level and of chronic respiratory

TABLE 2-11. EFFECTS OF GAS COOKING ON RESPIRATORY ILLNESSES AND SYMPTOMS IN CHILDREN

Study Population	Outcome Measure	Results
BRITISH STUDIES:		
5758 children, 6 to 11 yrs, England and Scotland (Melia, 1977).	Major respiratory symptoms and diseases individually, and as a single composite variable describing the presence of any 1 of 6 symptoms or diseases.	Significant associations gas between cooking and selected symptoms and diseases, and of a composite variable.
2408 children, 42% of original 5758 in above study (Melia et al., 1979).	Single composite variable as described above.	Relative risk for composite variable generally exceeded 1.0; risk varied and decreased with age.
4827 children, 5 to 11 yrs, England and Scotland (Melia et al., 1979).	Single composite variable as described above.	Significant effect of gas stoves on composite variable in urban areas only.
808 children, 6 to 7 yrs, United Kingdom, (Florey et al., 1979).	Single composite variable as described above.	Borderline significant association between composite variable and gas stoves. Increased prevalence as bedroom NO_2 levels increased in a sample with measurements (n = 80).
191 children, 5 to 6 yrs, England (Melia et al., 1982a,b).	Single composite variable as described above.	No significant association between bedroom NO_2 levels and prevalence of composite variable.
390 infants, 0 to 1 yrs, England (Melia et al., 1983).	Respiratory illnesses and symptoms requiring physician visits, assessed prospectively.	No association between gas stove use and respiratory illnesses and symptoms.
1565 infants, 0 to 1 yrs, England (Ogston et al., 1985).	Respiratory illnesses and hospitalizations assessed prospectively to 1 yr.	No significant association between illness or hospitalizations and use of gas for cooking.

(continued on following page)

TABLE 2-11. (continued)

Study Population	Outcome Measure	Results
OHIO STUDIES:		
441 upper-middle class families including 898 children under age 16 (Keller et al., 1979a).	Incidence of acute respiratory illness, determined by bi-weekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
120 families from first study, including 176 children under age 12 (Keller et al., 1979b).	Incidence of acute respiratory illness, determined by bi-weekly telephone calls and validated by home visits.	Respiratory illness incidence similar in homes using gas and electric stoves.
HARVARD AIR POLLUTION HEALTH STUDY:		
8120 children, 6 to 10 yrs, six U.S. cities (Speizer et al., 1980).	History of physician-diagnosed bronchitis, of serious respiratory illness before age 2, of respiratory illness in last year.	Significant association between current use of gas stove and history of respiratory illness before age 2 (Odds ratio = 1.23).
10,106 children, 6 to 10 yrs, six U.S. cities. Expansion of above study (Ware et al., 1984).	Same as above.	Odds ratio for history of respiratory illness before age 2 decreased to 1.12 ($p = 0.07$).
OTHER STUDIES:		
676 children, third and fourth grades, Arizona (Dodge, 1982).	Prevalence of asthma, wheeze, sputum, cough as determined by parent completed questionnaire.	Significant association between use of gas stove and prevalence of cough (prevalence rate ratio = 1.97).
4071 children, 5 to 14 yrs, Pennsylvania (Schenker et al., 1983).	Major respiratory illnesses and symptoms as determined by parent-completed questionnaires.	No significant association between use of gas stove and any symptom or illness variable.

(continued on following page)

TABLE 2-11. (continued)

Study Population	Outcome Measure	Results
1138 children, 6 to 12 yrs, Iowa (Ekwo et al., 1983).	Major respiratory symptoms and illnesses as determined by parent-completed questionnaires.	Significant association between current gas stove use and hospitalization for respiratory illness before age 2 (Odds ratio = 2.4).
121 children, 0 to 13 yrs, Connecticut (Berwick et al., 1984).	Number of days of illness.	Number of days of illness associated with average hours of heater use.
231 children, 6 yrs., Netherlands (Hoek et al., 1984).	Comparison between NO_2 levels in homes of cases (children with asthma) and controls.	NO_2 distributions similar in homes of cases and controls.

symptoms have not shown consistent adverse effects of gas stoves (Comstock et al., 1981; Jones et al., 1983; Fischer et al., 1985) (Table 2-13).

Definitive statements concerning the health risks of indoor NO_2 exposure cannot be made at present. Many studies have examined respiratory illnesses, respiratory symptoms, and lung function in children and adults, but their results are not consistent and are not adequate for establishing a causal relationship. Variations in the characteristics of the study populations and differing end points may partially explain the differences among the studies. NO_2 exposures were directly measured in only a few of the studies. Surrogate measures of NO_2 exposures, such as stove type, do not accurately classify the exposures of individual subjects. As a result, the studies that have used such surrogates may have been biased toward not detecting an effect.

2.2.4.4 Summary of Knowns and Unknowns for NO_2. Since NO_2 is a NAAQS pollutant, it is the subject of study under other portions of ORD research program. Research needs to address major uncertainties in health risk assessment are multiple and are separately documented by CASAC and ORD. Therefore, these uncertainties and research needs will not be repeated here. Rather, the discussion will focus on certainties and uncertainties regarding NO_2 only in the context of indoor air. These issues are not duplicative of those relating to outdoor air.

TABLE 2-12. EFFECTS OF GAS COOKING ON LUNG FUNCTION IN CHILDREN

Study Population	Lung Function Measure	Results
808 children, 6 to 7 yrs, United Kingdom (Florey et al., 1979).	PEFR, $FEV_{0.75}$ FEF_{25-75}	No association with NO_2 levels or presence of gas stove.
898 children, 0 to 15 yrs, from 441 families, Ohio (Keller et al., 1979a,b).	FVC, $FEV_{0.75}$	Data on children not presented separately. No association with presence of a gas stove.
8120 children, 6 to 10 yrs, six U.S. cities (Speizer et al., 1980).	FVC, FEV_1	Overall reduction of 16 ml and 18 ml respectively, for FEV_1 and FVC in children from homes with gas stoves.
16,689 children, 6 to 13 yrs, 7 areas in U.S. (Hasselblad et al., 1981).	$FEV_{0.75}$	Significant reduction of 19 ml associated with gas stove use in older girls only.
676 children, 9 to 11 yrs Arizona (Dodge, 1982).	FEV_1	No effect of gas stoves on pulmonary level or rate of growth.
183 children, 6 to 12 yrs, Iowa (Ekwo et al., 1983).	FEV_1, FEF_{75}, FEF_{25-75}	No change after isoproterenol challenge in children from homes with gas stoves.
9720 children, 6 to 10 yrs, six U.S. cities (Ware et al., 1984).	FEV_1, FVC in FEV_1,	Significant reduction of 0.6% and FVC of 0.7%. Not significant after adjustment for parental education.
3175 children, 5 to 14 yrs, Pennsylvania (Vedal et al., 1984).	FVC, $FEV_{0.75}$, FEF_{25-75}, $Vmax_{75}$, $Vmax_{90}$	No association with use of gas stove.

TABLE 2-13. EFFECTS OF GAS COOKING ON PULMONARY ILLNESS, SYMPTOMS, AND FUNCTION OF ADULTS

Study Population	Outcome Measure	Results
441 upper-middle class families, including 1054 adults over 15 yrs, Ohio (Keller et al., 1979a).	Incidence of acute respiratory illness, determined by biweekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
120 families from first study, including 269 adults over 18 yrs, Ohio (Keller et al., 1979b).	Incidence of acute respiratory illness, determined by biweekly telephone calls and validated by home visit.	Respiratory illness incidence similar in homes with gas and electric stoves.
1724 adults, ages >20 yrs, Maryland (Comstock et al., 1981).	Major chronic respiratory symptoms, FEV_1, FVC.	Association between gas stove use and increased prevalence of respiratory symptoms, FEV_1 <80% predicted, FEV_1/FVC <70%, found in nonsmoking males only.
708 adults, ages >20 yrs. Nonsmoking sample of above population (Helsing et al., 1982).	Major chronic respiratory symptoms, FEV_1, FVC.	Significant association between gas stove use and increased prevalence of chronic cough and phlegm, low FEV_1/FVC.
102 nonsmoking women in lowest quartile of FEV_1 compared to 103 nonsmoking women in highest quartile, Michigan (Jones et al., 1983).	Comparison of proportions of cases and and controls currently using gas stoves.	Marginal association between use of gas stove and lower lung function, (odds ratio = 1.8, p = 0.08).
97 nonsmoking adult females, Netherlands (Fischer et al., 1985).	IVC, FEV, FVC, PEF, MEF_{75}, MEF_{25}, MMEF.	Cross-sectional analysis showed an association between current NO_2 exposure and decreases in most pulmonary function measures. No association with longitudinal decline in pulmonary function.

Major knowns:

1. NO_2 levels observed in some homes can decrease pulmonary function in asthmatics.

2. As shown in animal toxicology studies, exposure to NO_2 can cause effects on lung host defenses, biochemistry, function, and structure, and some of the effects are likely to be irreversible. Chronic exposure is a significant cause for concern.

3. Epidemiologic evidence is sufficient to suggest that exposure to emissions from gas stoves can cause effects on the pulmonary systems of children and perhaps adults.

4. NO_2 is likely to have additive or synergistic effects with other indoor pollutants.

Major unknowns:

1. Except for NO_2 associated with gas stoves, the effects of NO_2 in combination with other indoor pollutants are totally unknown. Given the nature of the toxicity of NO_2, this is a substantial information gap. Even if there were far more knowledge of the effects of NO_2 alone, there would still be a need to assess NO_2's contribution to the effects in a mixture to enable assessment of an NO_2 source and development of an effective mitigation strategy.

2. Explicit information on the number of different susceptible subpopulations is unavailable.

3. As a whole, the epidemiologic studies on the effects of cooking with gas stoves are not conclusive. Most of the quantitative exposure assessment was not adequate in these studies. Other elements of the study design also contribute to the lack of clear conclusions. The causative agent in these studies is also not definitive. It is assumed, for sound reasons (U.S. Environmental Protection Agency, 1982b), to be NO_2, but other chemicals are emitted, making additive and perhaps even synergistic effects likely.

2.2.5 Sulfur Dioxide

2.2.5.1 Monitoring. Many measurements have been made of indoor versus outdoor particulate matter, as well as organic and inorganic compounds associated with particulate matter, to obtain a meaningful relationship between the two (van Houdt et al., 1984; Alzona et al., 1979; Yocom et al., 1970). In general, indoor-to-outdoor ratios (I/O) are close to unity for inhalable particulate matter (IPM) or respirable particulate matter (RPM) when no smokers are present in the home. By contrast, I/O ratios are generally less than unity when total suspended particulate matter is involved due to the filtering action of crevices (Yocom et al., 1982; Yamanaka and Maruoka, 1984; Cohen and Cohen, 1980).

Indoor SO_2 will invariably be approximately 30 percent less than the outdoor values due to both the preponderance of outdoor sources, and the chemical reactivity of SO_2 with interior surfaces and ammonia generated by humans and animals in the indoor microenvironment. In the special case only, where one burns kerosene indoors in an unvented device using a poor grade of fuel that contains sulfur, will SO_2 be generated indoors, and will indoor values exceed outdoor values. Table 2-14 reports results of concentrations measured in such homes by Leaderer et al. (1984). These results indicate that people in such homes may be exposed to levels of SO_2 that may be of concern for susceptible individuals such as infants or the elderly patient with respiratory distress.

2.2.5.2 Health Effects. The health effects associated with exposure to SO_2 in combination with particles are discussed in Section 2.3.2.4. Reports of the effects of exposure to SO_2 alone are discussed here. Exposure of animals to levels of SO_2 <1 ppm have shown only decrements in pulmonary function (U.S. Environmental Protection Agency, 1982a).

Asthmatic subjects are at least one order of magnitude more sensitive to SO_2 inhalation than are otherwise normal individuals (U.S. Environmental Protection Agency, 1982a, 1986a). Mildly asthmatic subjects have been studied, and it has been observed that approximately 50 percent of the asthmatic volunteers experience at least a doubling of airway resistance at concentrations at or below 0.75 ppm SO_2 (Roger et al., 1985; Horstman et al., 1986). In addition, asthmatic subjects have been studied following very short exposure to SO_2, and significant responses have been measured following exposures of only one minute duration. Other health effects associated with exposure to SO_2 are discussed in Section 2.3.2.4, with the discussion of particulate matter.

2.3 PARTICLES AND OTHER COMBUSTION PRODUCTS

2.3.1 Introduction

The term particulate matter represents a broad class of substances both chemically and physically. It consists of liquids, aerosols, or solid particles capable of suspension in air. Airborne particles exist in diverse sizes and compositions that can vary widely under changing influences of source

TABLE 2-14. TWO-WEEK AVERAGE SO_2 LEVELS BY LOCATION FOR HOMES IN SIX PRINCIPAL SOURCE CATEGORIES*

Source Category Location	SO_2 ($\mu g/m^3$)			
	N	Mean	SD	% >$80 \mu g/m^3$
NO K** Heater or Gas Stove				
Outdoors	-	-	-	-
House Average	11	0.6	2.0	0
Kitchen	-	-	-	-
Living Room	12	1.1	3.7	0
Bedroom	13	1.7	4.2	0
One K Heater, No Gas Stove				
Outdoors	-	-	-	-
House Average	25	68.4	86.8	24.0
Kitchen	-	-	-	-
Living Room	25	72.4	92.3	28.0
Bedroom	25	62.9	98.0	20.0
No K Heater, Gas Stove				
Outdoors	-	-	-	-
House Average	5	0.5	0.7	0
Kitchen	-	-	-	-
Living Room	5	1.1	1.5	0
Bedroom	5	0.0	0.0	0
One K Heater, Gas Stove				
Outdoors	-	-	-	-
House Average	3	89.9	91.2	33.3
Kitchen	-	-	-	-
Living Room	3	45.6	57.3	33.3
Bedroom	3	134.1	179.4	33.3
Two K Heaters, No Gas Stove				
Outdoors	-	-	-	-
House Average	5	120.4	66.4	40.0
Kitchen	-	-	-	-
Living Room	5	90.1	62.4	60.0
Bedroom	5	150.7	146.5	60.0
Two K Heaters, Gas Stove				
Outdoors	-	-	-	-
House Average	2	110.0	81.5	50.0
Kitchen	-	-	-	-
Living Room	2	118.0	102.4	50.0
Bedroom	2	101.9	60.7	50.0

*Repeat monitors period data (n = 19) are included.
Samples were lost for two residences; one residence the monitors were capped early by the residents and for the 2nd residence repeated efforts by the interviewers to retrieve the monitors failed.

**K = kerosene

Source: Leaderer (1984a).

contributions and interactive conditions (U.S. Environmental Protection Agency, 1982a). In regard to size, airborne particles tend to cluster in two groups: coarse particles, generally larger than 2 to 3 μm in diameter; and fine particles, generally smaller than 2 to 3 μm in diameter. Chemical composition is less definable due to interactions among particles and other pollutants, although photochemical oxidation plays a negligible role in transforming chemicals indoors. Some particles are highly reactive (e.g., acidic or basic) with other pollutants or with biological systems and indoor materials. Condensations from particle-particle, or gas-particle interactions do still occur. While the relationship between chemical type and particle size has been relatively well characterized for ambient air (the fine mode consisting primarily of sulfates, organics, ammonium, nitrates, carbon, lead, and other trace metal constituents; the coarse mode consisting of silicon components, iron, aluminum, sea salt, and plant particles, with some overlap between fine and coarse modes) characterization of indoor particles is less well defined (U.S. Environmental Protection Agency, 1982a).

2.3.2 Particles and Organics from Combustion

Combustion sources (combustion appliances and tobacco smoking) are probably the chief indoor generators of fine-mode particles which contain a host of organic and inorganic material about which little is known. Spray and cooking aerosols may also contribute to the total fraction of fine mode particles. Biological contaminants, including viruses, bacteria, fungal spores and fragments, pollens, fragments of house dust-mite feces, and dried, reentrained animal secretions (e.g., urine, saliva) and animal dander, may also be found primarily in the fine-mode fraction. Coarse-mode fractions which may consist largely of material carried in from outdoors such as dusts, or larger biological fragments such as mold parts or insect fragments, and which may settle on floors and carpeting and be reentrained through human activity, have not been well-characterized.

It has been determined that the I/O ratios are close to unity for IPM or RPM when no smokers are present. In contrast, I/O ratios are generally less than unity when total suspended particulate matter is involved due to the filtering action of the dwelling's shell (Yocom et al., 1982; Yamanaka and Maruoka, 1984; Cohen and Cohen, 1980).

2.3.2.1 Occurrence and Sources.

2.3.2.1.1 Unvented kerosene space heaters. Emission rates for particles generated by combustion from kerosene space heaters are reported in Table 2-15.

A study conducted by Yamanaka and Maruoka (1984) near the city of Kyoto, Japan, examined the mutagenicity of the extract recovered from airborne particles inside and outside a home with unvented kerosene heaters. Samples were collected over a one-month period inside and outside the home without a smoker, and were examined for mutagenicity using the Ames bioassay techniques. Mutagenic activity on the order of two- to threefold greater was found in indoor compared to the outdoor samples, suggesting that this increase in mutagenicity was a consequence of the kerosene heater use.

TABLE 2-15. PARTICULATE EMISSION RATES FROM KEROSENE SPACE HEATERS[a]

Radiant (7760 Btu/hr)	Emission Rate (mg/hr)
New Unit, Normal Flame	0.16
New Unit, Low Flame	0.19
Old Unit, Normal Flame	0.13
Convective (7430 Btu/hr)	
New Unit, Normal Flame	<0.03
New Unit, Low Flame	<0.02
Old Unit, Normal Flame	0.034

[a] Mass of particles from 0.005 to 0.4 μm diameter determined by electrical mobility detector, particle density of 2.0 g/cm.

Source: Traynor et al. (1982a, 1983).

Traynor et al. (1986) measured particulate emissions of PAH and nitrated PAH from kerosene combustion. They used a radiant and a convective heater in a 27 m^3 environmental chamber operated at approximately 1.1 ach to collect particulate emissions on a XAD/filter collection device operated at 6.8 m/s.

Because the particulate emissions from a well tuned convective heater are negligible, it was necessary in this study to maladjust the convective heaters to force particulate production. The convective heaters were maladjusted by lifting the exterior shells approximately 1 cm to induce sooting. The well-tuned radiant heaters' emission rates were consistent with the published data.

The study reported a positive response in the bioassay analysis of the extract of both sets of heater filter, indicating the presence of nitrated PAH's. In addition, the radiant heater particulates were demonstrated to contain highly mutagenic dinitro-PAHs (Table 2-16). Particulate matter from the convective heater showed no such response (Traynor et al., 1986).

TABLE 2-16. NITRATED PAH SOURCE STRENGTHS FROM WELL-TUNED RADIANT AND MALADJUSTED CONVECTIVE KEROSENE SPACE HEATERS (ng/hr)

Compound	Radiant 1,2,4,5	Radiant 3	Maladjusted Convective 1,2
1-nitronaphthalene	280	140	380
9-nitroanthracene	--	56	--
3-nitroflouranthene	1.9	--	--
1-nitropyrene	47	8.2	

[a] All measurements represent total for XAD/filter extract except 1-nitropyrene which was filter extract only.

[b] Analytical detection limits for nitro-PAH was 1.0 ng/hr.

[c] Dinitropyrenes (1,3-DNP, 1,6-DNP, 1,8-DNP) were measured by other researchers using bioassay techniques to give a source strength of 0.2 ng/hr.

Source: Traynor et al. (1983).

2.3.2.1.2 Gas appliances. Gas appliances have been found to be a source of particulate matter (Gas Research Institute, 1985; Girman et al., 1982; Traynor et al., 1982b,c). Girman et al. (1982) reported that the average emission rate for particles less than 25 µm was 0.24 to 0.62 µg/kJ for gas stoves. GRI reported a value of 0.25 µg/kJ. The dominant element of RSP was reported by Traynor to be carbon (Traynor et al., 1982b,c).

Table 2-17 contains particulate emissions reported by Traynor et al. (1983).

Emissions of RSP from natural gas-fired appliances have been reported to be on the order of 4.6 mg/hr per burner for a gas stove, 0.1 mg/hr for a gas oven, and 0.2 to 3.2 mg/hr for unvented space heaters (Fisk et al., 1985). RSP resulted from the use of top burners rather than from the use of the ovens (U.S. Department of Energy, 1985).

TABLE 2-17. GAS RANGE AND OVEN PARTICULATE EMISSION RATES (mg/hr)

Gas-fired burner operating 16 min, Fuel input 8730 Btu/hr	
Average	4.6[a], 3.8[b]
Range	1.9 to 9.5[a]
	2.2 to 5.7[b]
Gas-fired oven at 180°C for 1 hr, Fuel input rate 7970 Btu/hr	
Average	0.126[a], <0.42[b]
Range	0.118 to 0.126[a]

[a] Particles (<0.5 μm) based on electrical mobility analyser; particle density assumed to be 2.0 g/cm.

[b] Particles (<2.5 μm) based on gravimetric analysis of filter catch.

Table 2-18 contains reported particulate emissions for gas-fired space heaters.

TABLE 2-18. PARTICULATE EMISSION RATES FOR A GAS-FIRED BLUE-FLAME SPACE HEATER

Test Conditions		Emissions (mg/hr)
Well-tuned, in laboratory, full input	Range	0.21 to 3.23
	Average	1.31
Well-tuned, in laboratory, partial input	Range	<0.026 to 0.0264
	Average	<0.93

Sexton and Repetto examined the particulate matter from cooking stoves and cigarette smoke. The mutagenic density as revertants per m^3 in *Salmonella typhimurium* strain TA98 was determined for the particulate extracts. The particle emissions from gas stoves were not found to be highly mutagenic (Sexton and Repetto, 1982).

2.3.2.1.3 *Wood-burning stoves and fireplaces*. In certain parts of the country, wood-burning stoves and fireplaces have a strong influence on ambient air quality and hence on the quality of the air entering the home. It is estimated that in the Pacific Northwest, up to 50 percent of the homes rely on wood fuel to provide at least a portion of their their heating needs. The impact of residential firewood use on air quality has been detailed by Lipfert (1982). In six large cities, the impact of wood burning on ambient total suspended particles (TSP), NO_x, CO, and BaP was significant. Woodsmoke from residential wood stoves can also be a prime source of mutagens in indoor air. It has been reported that the reaction of the wood smoke with NO_2 and O_3 greatly increases its mutagenic activity.

Particulate emissions from woodstoves and fireplaces vary widely, depending on the design and operation of the unit. A joint TVA/BPA study released in 1985 indicated that airtight and non-airtight wood heaters were a statistically significant source of indoor TSP and RSP. In addition, these combustion devices were found to contribute significant quantities of PAHs (Neulicht and Core, 1982; Hytonen et al., 1983; Moschandreas et al., 1981a). In general, airtight wood stoves and catalytic wood stoves contributed less pollution than the non-airtight units.

Samples of airborne particulates collected in a room which was alternately heated via electricity and woodburning were examined in the presence and absence of tobacco smoking. An airtight heater was found to cause only minor changes in the PAH concentration and the NO mutagenic activity. The most significant increase occurred when wood was burned in an open fireplace. There were notable mutagenic effects compared to those activities resulting from tobacco smoke (Alfheim and Ramdahl, 1984).

2.3.2.1.4 Attached garages. The potential impact on indoor air quality from this type of source is seen in a study by Vu Duc and co-workers (1981). They characterized motor exhausts in an underground car park using a Hi-Vol cascade impactor to determine the size distribution of the exhaust particulate. They reported that 60 percent of the lead and cadmium detected were located in the particles of submicron aerodynamic diameters and that 50 percent of the PAHs were absorbed on particles of less than 1.1 μm (Vu Duc and Favez, 1981). Since there are no significant indoor sources of lead, lead found in the indoor environment is generally attributed to vehicle exhaust (Winchester and Nelson, 1979; Yocom et al., 1982).

2.3.2.1.5 Conclusions. A summary table of emission rates for particulates from indoor combustion sources has been published by the U.S. Department of Energy (1985) and is presented in Table 2-19.

TABLE 2-19. EMISSION RATES FOR PARTICULATE AND PARTICULATE-BOUND MATERIALS (mg/hr)

Source	Appliance Type	Particles	Benzo(a)pyrene
Kerosene Space Heaters	Radiant	0.13-0.16	--
	Convective	<0.03-0.034	--
Gas Space Heaters		0.21-3.23	--
Wood Heaters		2.6	1.4 x 10E^{-5} 3.5 x 10E^{-3}
Gas Appliance	Range (1 burner)	1.9-30	--
	Oven	0.118-0.126	--

Source: U.S. Department of Energy (1985).

2.3.2.2 Exposure. The U.S. EPA's 1982 Criteria Document for Particulate Matter and Sulfur Oxides (U.S. Environmental Protection Agency, 1982a) and its first and second addenda (U.S. Environmental Protection Agency, 1982b, 1986a) review the scientific bases for the National Ambient Air Quality Standards (NAAQS) for particulate matter (PM) and SO_x. This assessment does not attempt to duplicate the references in the air quality criteria document but, instead, focuses on work, published after 1969, on total human exposure to air pollutants. The 1984 symposium sponsored by the Swedish Council for Building Research, Stockholm (Berglund et al., 1984), provides a broad background on the worldwide interest in indoor air pollution and human exposure.

The earliest data concerned with human exposure to indoor particles were more related to pesticides adsorbed on particles than to the particles themselves (Starr et al., 1974). Fugas (1975) used indoor monitoring networks to measure lead. This work showed some of the relationships of I/O concentrations in European towns. Shortly thereafter, Binder et al. (1976) determined that particulate dosage appeared to be caused more by exposure to indoor, rather than outdoor, pollutants.

At the same time, evidence began to emerge concerning the indoor pollutant load caused by cigarette smoking. Although the earliest work was more concerned with CO, PM from tobacco smoking quickly became an important area of scientific interest. Dockery and Spengler (1977), Repace and Lowrey (1980), Repace (1981), Bock (1982), Girman et al., (1982), Johnson et al. (1984), and many others demonstrated the widespread effect of smoking on human health, both alone and in conjunction with other air pollutants such as particulates, CO, and VOCs.

By conventional measures, the quality of ambient air has steadily improved over the years. However, although outdoor TSP concentrations may have decreased, human exposures to inhalable particulates, RSP, that is, below 2.5 μ in size, probably has increased due to concentrations of fine aerosols and ultrafine particulates generated within homes and offices. In addition, because most people spend more than 90 percent of their time indoors, they will be proportionally subjected to any elevated levels of pollutants from indoor sources. Modern studies tend to assume that central monitoring stations do not reflect or predict actual personal exposures. Continued research is needed on the relationships between ambient concentrations and actual exposure.

PM is produced by incomplete combustion and suspension of ashes even when combustion is complete (except for a properly tuned gas flame). Consequently, cigarette smoking, woodstoves, and kerosene heaters will generate combustion particles into the indoor microenvironment which add to the noncombustion particle loading. It is unfortunate that the measurement techniques routinely used in reported studies are unable to discriminate between the combustion particles and the other household particles generated from human activities and intrusion from the outside air. As described by Mage et al. (1985) the particle mass collected is an heterogeneous mixture of material, with chemical properties ranging from inert to highly toxic and carcinogenic. Thus, a mere measure of mass concentration in $\mu g/m^3$ has little direct relationship to a health effect, since the percentage of materials thought to be inert can range from 0 to 100 percent. Further evidence shows that much of the RSP in an indoor microenvironment is generated by human activity (e.g., particle resuspension when walking on carpets, shedding of hair and skin detritus, cooking, vacuuming, etc.) so that an average taken over a 24-hour period will smooth out the high periods of human exposure with the low periods of quiescent air when no one is home. Spengler et al. (1985) show that personal 24-hour RSP exposures are consistently higher than the time-weighted average concentration predicted by 24-hour indoor and outdoor values from stationary monitors.

Additional complications exist because all these data are fragmentary in time (24-hour averages), and they do not cover seasons which are known to introduce trends in the data due to variations in activity patterns and air exchange (air conditioning versus heating). Table 2-20 gives the range of indoor and outdoor RSP concentrations and the comparative personal exposures of the people living in the homes under study. It is interesting to note that at most percentiles the personal exposure exceeds the indoor microenvironment concentration; this cannot be credited to outdoor concentrations which are invariably lower than those indoors. Other indoor RSP data from Spengler et al. (1985) are also given in Table 2-20.

TABLE 2-20. QUANTILE DESCRIPTORS OF PERSONAL, INDOOR, AND OUTDOOR RSP CONCENTRATIONS, BY LOCATION

	RSP Sample		RSP Quantile, µg/m³						
City	Group	N	95%	75%	50%	25%	5%	Mean	SE
Kingston	personal	133	99	47	34	26	19	42	2.5
	indoor	138	110	47	31	20	10	42	3.5
	outdoor	40	28	22	16	12	6	17	2.7
Harriman	personal	93	122	54	35	24	15	47	4.8
	indoor	106	129	45	27	18	10	42	4.1
	outdoor	21	34	23	15	13	9	18	4.0
Total[a]	personal	249	113	48	34	26	17	44	2.8
	indoor	266	119	46	29	20	10	42	2.6
	outdoor	71	33	23	17	13	7	18	2.1

[a] Includes samples from 13 subjects living outside Kingston and Harriman Tennessee town limits and from four field personnel.

Source: Spengler et al. (1985).

2.3.2.3 Monitoring of PM. Particle samplers for outdoor ambient air monitoring are large, noisy and cumbersome to use. Some continuous inhaled particle (IP) monitors are available (e.g., the piezobalance), but also are too large, inaccurate or too complex for THE monitoring. The smaller commercial IP monitors (e.g., GCA Miniram) have poor sensitivities and/or unknown validity at the low particulate concentrations found in nonoccupational environments. None of the continuous (direct-reading) monitors will collect a physical sample for subsequent chemical analysis. An accurate size-selective sampler (under 10 mm) that also can provide direct-reading information is needed.

A small, unobtrusive version of the dichotomous sampler is needed for indoor microenvironmental measurements. A true particulate PEM that collects enough sample to accurately compare with microenvironmental measurements is also needed. While pilot studies on real populations are now feasible with collection-type PEMs for airborne IP matter, additional laboratory development of this type of PEM must be conducted before precise studies are possible.

The developmental efforts for exposure monitors for inhaled particulate matter have either been directed towards a true PEM or toward a portable sampler that can be carried easily from one microenvironment to another. Because of gaps in the technology, the latter approach has been pursued more often to provide an unobtrusive PEM that collects enough particulate matter for subsequent analyses. Fletcher (1984) reviewed many of the exposure samplers available. Most studies have compromised by accepting the potential errors associated with predicting particulate exposure from microenvironmental measurements, as compared to those induced by the precision problems of PEMS and the excessive burden of an obtrusive PEM.

A small, portable, single-channel sampler was designed for a joint effort between Harvard and the Electric Power Research Institute (EPRI) (Dockery and Spengler, 1981). It operates at 1.7 l/min and is provided with a 2.5-mm cyclone inlet. This sampler has been widely used and was utilized by Harvard in conjunction with an activity diary to relate the microenvironmental measurements to exposure. The low flowrate and unavailability of inlets with other cutpoint size are the major drawbacks to this sampler.

In an EPA-supported effort National Bureau of Standards (NBS) developed an alternative sampler (McKenzie et al., 1982) with real channel capabilities (2.5 and 10 µm D_{ae}) in a portable version similar in size to the Harvard/EPRI sampler. This NBS version collects a 0 to 2.5 µm and a 2.5 to 10 µm fraction, operates at 6 l/min, and is powered by a battery pack. The particle separation between the channels is geometric, and is achieved by using an 8-µm Nuclepore filter followed by an absolute filter. This is less desirable than an aerodynamic separator and requires that the coarse particle filter substrate be of polycarbonate material.

A developmental effort is currently under way by EPA to produce a higher flow rate PEM with interchangeable cutpoint inlets. Using the low current-drain pump and 2.5- and 10-µm inlets developed for a 10 l/min dual-channel microenvironment sampler in the Indoor Air Program (IAP), the effort is being directed toward a sampler that is minimally obtrusive. Sampling at 10 l/min should substantially improve the analytical capabilities and possibly permit sampling times as short as 12 hours.

Nongravimetric samplers, which use techniques such as light scattering and piezoelectric crystal frequency shift, are also available commercially. These samplers have proven useful in screening studies, but all have significant technical limitations, such as poor sensitivities or interference problems. Their utility as exposure monitors, either as PEMs or microenvironment samplers, has not been adequately demonstrated.

Table 2-21 summarizes the mass collected (at 100 percent efficiency) by several commonly used particulate samplers for various sampling times and flow rates.

2.3.2.4 Health Effects. The U.S. Environmental Protection Agency (1982a, 1986b) has extensively reviewed the health effects from exposure to particles. The most extensive data are derived from epidemiological studies of outdoor air. In these studies, the exposure assessments were limited primarily to measurement of the mass of PM; only rarely was the mass characterized chemically. Additional supporting data from animal toxicological and human clinical studies are chemical-specific. Careful evaluation of the entire data base indicates that PM causes adverse health effects. However, relating this entire data base to the indoor situation is not scientifically possible, inasmuch as specific chemicals are different indoors and outdoors, and for the most part specific chemicals in PM have not been characterized indoors or outdoors.

However, there are chemical-specific elements of the entire PM data base that can be useful in assessing the potential effects of exposure to indoor PM. Examples of useful data base elements include: ETS (discussed in a separate section), soot, particle-bound PAHs, and other combustion particles.

The respiratory system is the major route of human exposure to particles, and its structure and function, along with the physical and aerodynamic properties of the particles, determine where and how particles are deposited, retained, or cleared. Breathing patterns, as they relate to route and ventilation level, greatly influence where the deposition of inhaled particles occurs. The aerodynamic diameter (D_{ae}) generally has been characterized into two modes by EPA, the fine mode (<2.5 µm) and the coarse mode (>2.5 µm to ~15 µm, or greater). These distinctions relate to particle deposition, with fine-mode and

TABLE 2-21. QUANTITY OF PARTICLES COLLECTED IN MICROGRAMS AT A CONCENTRATION OF 30 $\mu g/m^3$

Sampler	Type[a]	Flow rate l/m	SAMPLING PERIOD (hr) 1	3	6	12	24	168
Harvard/ EPRI	PEM	1.8	3	10	19	39	78	544
Harvard Aerosol Impactor	μE	4	7	43	86	86	173	1,210
NBS Prototype	μE	6	11	32	65	130	259	1,814
EPA Prototype[b]	μE	10	18	54	108	216	432	3,024
Dichotomous[b] Sampler	A/μE	16.7	30	90	180	360	720	5,050
1-CFM Andersen Impactor	A/μE	28.3	51	153	306	612	1,223	8,564
4-CFM Andersen	A/μE	113	204	612	1,224	2,448	4,896	34,262
8-CFM Battelle (Prototype)	μE	226	408	1,224	2,448	4,896	9,792	68,524
PM$_{10}$HI VOL	A	1,133	2,039	6,118	12,236	24,473	48,946	342,619

[a] PEM - Personal (portable) exposure monitor
μE - Microenvironment sampler
A - Ambient

[b] Sample divided between 2 or more substrates

Source: Rodes (1986).

small coarse-mode particles depositing principally in the thoracic (tracheobronchial and pulmonary) region and larger coarse-mode particles depositing primarily in the extrathoracic (nasopharyngeal) region during nose-breathing. Oronasal breathing associated with minute ventilations exceeding 0.35 l/min can significantly alter deposition patterns (U.S. Environmental Protection Agency, 1982a).

The Air Quality Criteria for Particulate Matter and Sulfur Oxides document (U.S. Environmental Protection Agency, 1982a) lists a number of possibilities of adverse effects from inhaled particles. Included are possible irritant effects resulting in decreased air flow as a result of airway constriction, altered mucociliary transport, and changes in alveolar macrophage activity. These effects apply across a wide range of inhaled particles, acting alone, or in concert with common gaseous air pollutants, such as SO_2, NO_x, or ozone. Other toxic effects are more chemical-specific and, depending on the nature of the chemical, may include organs outside of the respiratory tract.

Bronchoconstriction, arising from chemical and/or mechanical stimulation of irritant neural receptors in the bronchi, has been reported as a response to short-term exposure to high levels of various inert dusts, as well as to acid and alkaline aerosols. Neurological receptors tend to concentrate near airway bifurcations, where particle deposition is greatest, so that stimulation may result in pulmonary reflexes, such as bronchoconstriction and coughing. These reflex actions may be related to the effects observed in various epidemiological studies, such as aggravation of chronic respiratory disease states including asthma, bronchitis, and emphysema. Individuals with asthma or emphysema and other respiratory diseases may have increased particulate deposition due to altered breathing patterns or airway structural changes, which may then contribute in a cascading effect to even more bronchoconstriction and particulate deposition (U.S. Environmental Protection Agency, 1986a).

New information discussed in the second addendum to the 1982 Air Quality Criteria for Particulate Matter and Sulfur Oxides document (U.S. Environmental Protection Agency, 1986a) supports the conclusions of the earlier document. Newly available studies that classify thoracic deposition and clearance of large particles are presented; deposition during oronasal breathing and deposition in possibly susceptible subpopulations such as children are assessed, and new information that could lead to health effects data refinement or reinterpretation are evaluated.

Tables 2-22 and 2-23 summarize the quantitative health data derived from epidemiologic studies reviewed in the second addendum to the 1982 criteria document on PM and SO_x (U.S. Environmental Protection Agency, 1986a).

It should be emphasized that the health data obtained for PM are generally the result of epidemiologic studies that correlate health effects observations in an exposed population with exposure to ambient air pollutant levels. The

TABLE 2-22. SUMMARY OF KEY QUANTITATIVE CONCLUSIONS BASED ON NEWLY AVAILABLE EPIDEMIOLOGICAL STUDIES OR ANALYSES RELATING HEALTH EFFECTS TO ACUTE EXPOSURE TO AMBIENT AIR LEVELS OF SO_2 AND/OR PM

Type of Study	Results Obtained	24-hr average pollutant level ($\mu g/m^3$)			Reference
		BS*	TSP	SO_2	
Mortality	Indications of increased mortality during London winters of 1958-59 to 1971-72, with most marked SO_2 effects evident at ~700-750 $\mu g/m^3$ and indications of small increases at BS levels <500 $\mu g/m^3$ and possibly as low as 150-300 $\mu g/m^3$.	<500	--	~700-750	Mazumdar et al. (1982)
	New analyses of same 1958-59 to 1971-72 London winter mortality data indicative of increased mortality at BS levels <500 $\mu g/m^3$ and no evident threshold at 150 $\mu g/m^3$.	<150-500	--	--	Ostro (1984)
	Unpublished reanalysis of same 14 year London mortality data using spectral transform multiple regression analyses confirming significant associations for total, cardiovascular and respiratory mortality, accounting for autocorrelation and temperature. Suggestion of more pronounced effects with 7-21 day cycles of exposure.	<500	--	--	Shumway et al. (1983)
	Unpublished reanalysis of same 14 year London mortality data using regression analyses that detrended data for time series autocorrelation, humidity, and temperature indicating significant associations between mortality and BS to below 100 $\mu g/m^3$, but not for SO_2 at <500 $\mu g/m^3$.	Continuous association from lowest (<100 $\mu g/m^3$) BS levels	--	>500	Schwartz and Marcus (1986)
Morbidity	Evidence for reversible (~2-3 wk) small (2-3%), but statistically significant decrements in FVC of school children following episodes in Steubenville, Ohio when 24-h TSP and SO_2 levels respectively ranged up to 220-420 and 280-460 $\mu g/m^3$, but not after "sham" episode with TSP = 160 and SO_2 = 190 $\mu g/m^3$. Larger decrements seen in subset of children.	--	220-420	280-460	Dockery et al. (1982)
	Evidence for reversible (~2-3 wk) small (3-5%), but statistically significant decrements in pulmonary function measures (FVC, $FEV_{1.0}$ MEF) for school children in the Netherlands during and after pollution episode when 24-h TSP, RSP, and SO_2 levels ranged up to 200-250 $\mu g/m^3$, but no effect shortly after day when same pollutants averaged 100-150 $\mu g/m^3$.	--	200-250	200-250	Dassen et al. (1986)

Source: U.S. Environmental Protection Agency (1986a).

TABLE 2-23. SUMMARY OF KEY QUANTITATIVE CONCLUSIONS BASED ON NEWLY AVAILABLE EPIDEMIOLOGICAL STUDIES RELATING HUMAN HEALTH EFFECTS TO LONG-TERM EXPOSURES OF SO_2 AND/OR PM

Type of Study	Results Obtained	Annual-Average Pollutants Levels ($\mu g/m^3$)		Reference
		TSP	SO_2	
Initial Cross-Sectional Analyses of Ongoing Longitudinal Study	Increased rates of cough, bronchitis and lower respiratory disease (in the absence of lung function changes) among school children in 6 U.S. cities significantly associated with annual-average TSP levels across range of approximately 30 to 150 $\mu g/m^3$ when analyzed for between city effects but not in relation to PM gradients within individual cities. Effects most clear for highest PM areas (~60-150 $\mu g/m^3$) versus lowest (~40-60 $\mu g/m^3$). No significant association with SO_2 except for cough.	~60-150	--	Ware et al. (1986)
Cross-Sectional Study	Significantly increased rates of persistent cough and phlegm (PCP) among young adults associated with annual-average SO_2 $\cong$115 $\mu g/m^3$ in highest exposure Utah community versus 3 lower exposure towns with SO_2 in 11-36 $\mu g/m^3$ range. No TSP gradient across four communities. Effects possibly due to intermittant high SO_2 peaks.	--	115	Chapman et al. (1985)
Longitudinal Study in Southwestern U.S. towns	Significantly increased prevalence of cough among children from highest pollution area (annual average SO_2 = 103 $\mu g/m^3$; intermittant 3 h peaks often exceeded 2,500 $\mu g/m^3$ or ~1 ppm) in comparison to lower pollution towns (annual SO_2 = 14 $\mu g/m^3$). No TSP gradient across high and low pollution towns. Effects possibly due to intermittant high SO_2 peaks.	--	103	Dodge et al. (1985)

*BS = British Smoke Method

Source: U.S. Environmental Protection Agency (1986a).

studies do not specifically involve measurement of indoor particles and, hence, do not assess effects from indoor air pollution. Furthermore, extrapolation of health effects from epidemiologic studies of ambient particle pollution should be approached with great caution because sampling methods for particles outdoors do not correlate with one another and often cannot be directly compared with the methods developed for indoor particle sampling.

Particles studied indoors have primarily been those in the fine-mode, primarily resulting from cigarette smoking, or as emissions from combustion appliances. Coarse-mode particles resulting from reentrainment of fibers, dust, house dust mite fecal pellets, animal and human dander, mold spores and fragments, probably constitute the second most common form of indoor particle pollution. Methods for routine sampling of many of these types of particles have not been developed, and their health impact is, for the most part, unknown.

2.3.2.4.1 Health effects associated with exposure to soot. Virtually every combustion process in which carbonaceous fuels are burned results in the production of soot. Soot often consists of clusters of very small (~30 nm) spheroids fused together to form particles with a 0.1 to 0.2 μ diameter. These soot particles contain an organic fraction (solvent extractable) and an elemental carbon fraction which appears to be similar to graphite. The combustion of diesel fuel, coal, fuel oil, and wood has been shown to produce soot particles with extractable organic matter that is mutagenic in short-term bioassays and, in several of these sources, has been shown to be tumorigenic in animals. Chemical characterization of these organics shows that they contain carcinogenic PAHs, methylated PAHs, nitrated PAHs, and oxidized PAHs.

Tokiwa et al. (1985) reported the presence of highly mutagenic and carcinogenic dinitropyrenes in the emissions of kerosene heaters, gas burners, and liquefied petroleum gas burners. The organic extracts of these emission particles showed mutagenic activity in the Ames Salmonella mutation assay in strains TA98 (1.5 to 5.3 revertants/μg) and TA97 (2.0 to 6.0 revertants/μg) without metabolic activation of S9. The organic extracts from kerosene heater emissions also showed positive in TA1538 and TA100 in the absence of S9. More mutagenic activity was found in the emission particles collected in the first 20 minutes of burning.

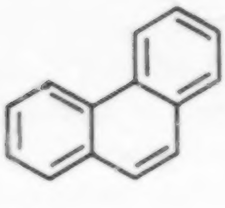
Higher mutagenic activity has been reported in samples extracted from air in homes that burn wood than from those that do not (Morin, 1985). Previous studies by Lewtas (1985) have shown that the organic extracts of the emission particles collected after a dilution tunnel from the air-tight wood stove were mutagenic in the Ames Salmonella mutation assay (1.3 revertants/μg for pine, and 0.93 revertants/μg for oak) in strain TA98 with metabolic activation of S9 mitochondrial fraction. The organic extract of the wood combustion emission using pine also showed dose-response skin tumor initiation activity in Sencar female mice in a 6-month study (Lewtas et al., unpublished data).

Occupational studies have shown the increased risk of lung, larynx, and skin cancer after exposure to coal soot, and increased incidence of scrotum and bladder cancer after exposure to coal tar in gashouse workers, stokers, asphalt, coal tar and pitch workers, coke-oven workers, and chimney sweeps (Cole and Goldman, 1975). Nonoccupation-related lung cancer has been linked to the exposure of indoor unvented coal combustion emissions in a study conducted in a rural county, Xuan Wei, located in Yunnan province in China (Mumford et al., 1987). The Xuan Wei residents have been exposed domestically to smoke from unvented coal and wood combustion. The county has unusually high lung cancer mortality rates that cannot be attributed to tobacco use or occupational exposure. The Xuan Wei women, mostly nonsmokers, have the highest lung cancer rates in China, and men's rates are among the highest. Lung cancer mortality rates are associated with the usage of smoky coal (comparable to U.S. medium-volatile bituminous coal) but not associated with the usage of wood. This is in agreement with the reports by DeKoning et al. (1984), who indicated no association of lung cancer with domestic burning of biomass (including wood, crop residues, and dung) in other rural areas of developing countries.

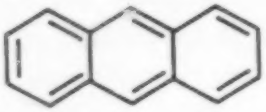
Studies of the noncarcinogenic effects of soot are relatively rare and consist primarily of animal inhalation toxicology studies of fly ash that was collected, stored, and reaerosolized (U.S. Environmental Protection Agency, 1982a). Generally, these studies showed that fly ash is of low toxicity compared to other aerosols such as sulfuric acid and of metals. However, high levels (>1 mg/m^3) did cause several types of pulmonary effects in animals after prolonged exposure. High-level exposure (>5 mg/m^3) to diesel particles also results in pulmonary effects after chronic exposure (McClellan et al., 1986).

2.3.3 Polycyclic Aromatic Hydrocarbons

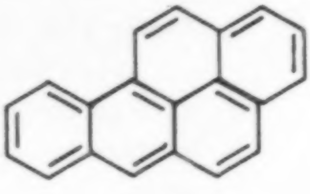
Polycyclic aromatic hydrocarbons (PAHs) represent a diverse group of combustion products whose common characteristic is a nucleus of five- or six-membered carbon rings in which interlinked rings have at least two carbon atoms in common (Zander, 1983). The number of rings varies from two to many. Configuration of ring structures determines physical and chemical properties and biological activities. Some examples of simple PAHs are depicted below.



phenanthrene



anthracene



benzo(a)pyrene

The PAHs are of specific human health concern because many of them are procarcinogenic, cocarcinogenic, or carcinogenic, and they affect the immune and cardiovascular systems. They are breathed into the lung as volatiles or they can adsorb to the surface of deposited particles and be inhaled.

2.3.4 Other Combustion Organics

PAHs can form many substituted compounds, which also may be pro-, co-, or frankly carcinogenic. In addition, combustion products include heterocyclic compounds, as well as compounds of nearly every class known, including aldehydes, ketones, nonpolycyclic hydrocarbons, aromatics, organic acids, and nitrosamines, among others.

2.3.5 Interaction of Particulate Matter and Organics

Beyond the specific issues discussed above, one of the concerns for both carcinogenic and noncarcinogenic effects of PM is related to the organics bound to the particles. The particles and bound organics have properties that influence deposition, retention, and bioavailability. Hence, the specific chemical-physical characteristics of the particles and organics have major influence on the health effects. One of the most classical examples is that BaP alone has low carcinogenic potency; ferric oxide alone is not a carcinogen. When BaP is coprecipitated with ferric oxide and instilled into the lungs of animals, cancers result. Many particles adsorb organics as they are created in combustion processes. However, once within the air, they can adsorb other materials (i.e., radon, pesticides, other organics from home products). These absorbed materials will have different pulmonary deposition sites and deposition efficiencies when adsorbed on a particle, as opposed to existing in vapor phase. Retention and pharmacokinetics would also be affected. Thus, even an inert particle could contribute (in a currently unknown way) to health risks from indoor air. Assessment of the risks of individual pollutants does not take into account such complex interactions. Risk assessments on complex mixtures must be done if a clear picture of the health effects of indoor pollutants is to emerge.

2.3.6 Monitoring

Concentrations of polynuclear aromatic hydrocarbons (PAHs) and related compounds, like those of the volatile organics (Pellizzari et al., 1982; Wallace, 1986b), have often been found to be higher indoors than outdoors (Chuang et al., 1987; Wilson et al., 1985; National Research Council, 1986c). Because many of these compounds are known carcinogens, and because many more show potential carcinogenicity (as evidenced by mutagenicity or other bioassays) exposure to them is of major concern. Sources of PAHs in the indoor environment include combustion devices, such as furnaces, woodstoves, fireplaces, and kerosene heaters. Additional contributions to the PAH levels can come from personal activities, which include cooking and especially smoking. There may also be minor contributions from other sources, such as building materials, furnishings, polishes, and waxes.

Because the PAHs and other SVOCs are distributed in air between the particulate and vapor phases, depending on the vapor pressures of the compounds, the nature of available adsorptive surfaces, temperature, and other environmental conditions, sampling systems for PAHs must collect both particles and vapor. Thus, a filter may be used to collect the particles with the filter followed in series by a vapor trap, containing a sorbent such as polyurethane foam (PUF) or XAD-2 resin to collect that portion of the PNAs that is in the gas phase (Chuang et al., 1987a,b; Wilson et al., 1985). Analytical procedures usually involve separate extraction of the filter and sorbent, with subsequent analysis by high pressure liquid chromatography (HPLC) or gas chromatography/mass spectroscopy (GC/MS). These traditional sampling techniques may, however, greatly disturb the phase distributions existing in the air at the time of sampling. Consequently, particulate-associated chemicals may be underestimated, while the vapor phase concentrations may be inflated. Equivalent results for total PAH concentrations (particles plus vapor) are obtained by pooling the filter and sorbent extracts prior to analysis or by analyzing the extracts separately and adding the results together. Pooling the extracts eliminates the possibility that an inaccurate assessment of phase distribution will be made and should improve analytical detection capabilities.

The utility of the sampling system just described was evaluated in a small indoor monitoring study performed in Columbus, Ohio in 1985 (Chuang et al., 1987b). A sampling head identical to those used in the PS-1 ambient air sampler (General Metal Works, Cleves, Ohio) was located indoors and connected by vacuum tubing through a window port to an outdoor pumping system. The sampling head contained a quartz fiber filter followed by a PUF adsorbent cartridge. Samples were obtained at a flow rate of 7 cfm (200 l/Min), which allowed collection of quantities sufficient for both chemical analysis and bioassay in eight hours, yet was low enough that only 5 to 10 percent of the total air volume in a given house was sampled. Analyses were performed on the sample extracts without cleanup, using chemical ionization GC/MS. The sample size was more than adequate for most of the 17 target PAHs, which were present in the 1 to 3 ng/m^3 range for large molecules, such as benzo(e)pyrene, and in the 20 to 150 ng/m^3 range for smaller molecules, such as phenanthrene and fluoranthene. To analyze for the two target nitro-PAHs, 1-nitropyrene and 2-nitrofluoranthene, which were present in the 10 to 100 pg/m^3 range, it was necessary to pool three 8-hr extracts and employ negative chemical ionization mass spectrometry. Sample sizes were barely adequate for microbioassay (Lewtas et al., 1987).

Subsequent studies have indicated the desirability of going to a lower flow rate, 1 to 2 cfm (28 to 57 l/min) to minimize perturbation of the indoor air environment by the sampling itself. These flow rates should still provide adequate sample for chemical analysis, but if bioassay is planned, sampling over longer time periods will be necessary to collect adequate material.

The adsorbent used in the indoor monitoring study described above was PUF. Good collection efficiencies and sample recoveries (>80 percent) were obtained for PAHs having three rings or more. More studies of the relative suitability of PUF and XAD-2 resin for the collection and quantification of PAHs (Chuang et al., 1987b) have shown that XAD-2 is preferable to PUF for sampling of PAHs with three rings or fewer, both because it adsorbs a greater fraction of the sample, and because a smaller fraction is lost by volatilization after collection. Sample storage in sealed containers at 20 to 60°C in the dark for periods up to thirty days does not appear to produce significant losses of most PAHs from XAD-2 or PUF. Similar storage of filters does not lead to significant sample losses, except for benzo(a)pyrene and cyclopenta (c,d)pyrene, which are gradually lost, presumably because of chemical reaction. The level of benzo(a)pyrene, for example, decreases in 30 days to about 40 percent of its initial value. Extraction and cold storage of the filter extracts soon after collection can minimize this problem. The sampling efficiencies, recoveries, and storage stabilities for collection media used to collect the substituted PNAs and other semivolatile compounds comprising the more polar constituents of air samples have not been thoroughly investigated.

Although the nonpolar fraction of air samples generally contains most of the PAHs, it only accounts for about one-third of the mutagenicity observed in bioassays. Other compounds, including some substituted and derivatized PNAs, are found in the polar fractions and must account for the remaining bioactivity. Many of these compounds have not been characterized chemically, and the analytical methods necessary for their separation, identification and quantification, have not been fully developed. Therefore, a great deal of methods development research remains to be done before confident assessments of exposure to these carcinogens and toxic chemicals can be made.

One of the most striking findings of the indoor air methods evaluation studies mentioned above (Wilson et al., 1985; Lewtas et al., 1987) was that of the variables--fireplace use, gas or electric heating system and appliances, air exchange rate, and cigarette smoking --the experimental variable that had the greatest effect on both PAH levels and on mutagenicity was smoking. Mainstream cigarette smoke and its effects have been well-documented in the literature. However, the chemical characteristics of sidestream smoke and the associated exposures have not been as thoroughly studied. Many research needs are apparent (National Research Council, 1986c). The needs of exposure measurement methods include reliable methods for collection and quantification of nicotine, which can serve as a marker for tobacco smoke gas phase constituents, identification of marker compounds for the particulate phase constituents, and development of sampling and analytical methodology for these markers. Analytical methodology for the more polar PAHs and other semivolatile compounds associated with ETS must be developed or improved.

To ascertain true exposure to the PAHs and other semivolatile compounds in indoor and ambient air, it is necessary to know whether they are in the gas phase or are associated with particles, so that the particle size distribution determines to a large extent whether they are retained in the body. Few careful and accurate studies of the phase distribution of these compounds have been done (National Research Council, 1986c). Development and evaluation of sampling methodology that will quantitatively separate the vapor and particles, and that will maintain the integrity of the separately collected portions of the samples, is necessary. Preliminary work on denuder-based samplers to determine phase distributions has been done (Coutant et al., 1987), but further development is needed. Since sampler design affects the subsequent methods of analysis further analytical methods development for studies of the phase distribution of PNAs and related compounds is implied.

2.3.7 Woodsmoke

Woodsmoke is made up of a complex mixture of compounds, including aldehydes, such as acrolein, and PAHs, many of which are mutagenic. Some studies from developing countries indicate an association between high-level smoke exposure (i.e., relative to the U.S.) in dwellings and chronic pulmonary disease.

2.3.7.1 Health Effects. Some studies from developing countries indicate an association between intense smoke exposure in dwellings and chronic pulmonary disease. It should be noted that the following studies describe effects from smoke exposure in unventilated dwellings, so that concentrations are orders of magnitude greater than those expected indoors in the U.S. In a house-to-house survey of adults in a village in Nepal, Pandey (1984a,b,c) found that the prevalence of chronic bronchitis increased with the extent of domestic smoke exposure, as measured by the number of hours spent daily near the stove. In a subsequent study, Pandey et al. (1985) evaluated respiratory function of 150 women ages 30 to 44 years from two rural villages in Nepal. Domestic smoke exposure appeared to adversely affect lung function of smokers but not of nonsmokers. Epidemiological and clinical studies involving adults in New Guinea have also suggested adverse effects of domestic smoke exposure (Master, 1974; Anderson, 1979).

Respiratory effects of woodsmoke have also been examined in children from these countries. Anderson (1978) studied 1650 children drawn from two contrasting New Guinea communities, one at sea level where wood was not burned indoors and one in the highlands where wood was commonly burned indoors. A cross-sectional survey showed no differences between the two groups on spirometric testing, by physical examination, or by clinical history. During a 30-week surveillance period involving some of the wood smoke-exposed children and an unexposed control group, Anderson (1978) did not find a consistent relationship between woodsmoke exposure and respiratory abnormalities. In contrast, Kossove (1982) reported that Zulu infants under 13 months of age with severe lower respiratory tract disease were twice as likely to have a history of daily heavy smoke exposure compared to unexposed control infants. It has been estimated that about one-half of the world's households cook and/or heat daily with biomass fuels (Smith, 1986). In most cases, the fuels are burned under unvented conditions. The major health effects from high exposures to biomass combustion products are chronic obstructive lung disease, heart diseases including cor pulmonale, and acute respiratory infection diseases which cause high infant mortality and low birth weights.

While these studies implicate domestic smoke exposure as a possible risk factor for the development of respiratory disease in developing nations, their results should not be generalized to developed nations, where exposures are generally much lower and biomass fuels are not widely used.

Data on health effects of residential wood combustion in the U.S. are sparse. Honicky et al. (1983) described the case of an infant who was repeatedly hospitalized for severe lower respiratory tract disease characterized by wheezing and pneumonia. The child's illnesses stopped when the parents removed the woodstove from the home. This case prompted Honicky and colleagues (1985) to conduct a prevalence study of respiratory symptoms in 62 Michigan children, 31 from homes with and 31 from homes without woodburning stoves. The proportion of children with moderate or severe symptoms during the previous winter was much greater in the group from homes with woodstoves: 84 percent had at least one severe symptom as compared to 3 percent of the control group. However, in a similar study in Massachusetts, Tuthill (1984) found no association between use of a woodburning stove and chronic respiratory disease, respiratory symptoms, and excess respiratory symptoms. Woodsmoke components were not measured in the subjects' homes in either investigation.

2.3.8 Major Knowns and Unknowns: Health Effects

This section only addresses the health effects of PM as they relate to indoor exposures. Thus, it does not include issues critical to outdoor PM. PM as part of ETS is discussed in Section 2.4.

Major Knowns:

1. There is a cancer potential associated with exposure to PM.
2. The relationship between exposure and dose of PM is heavily influenced by several factors including activity patterns, age of the person, and disease status of the person. Organics adsorbed onto particles also have a different dosimetry than the organics alone, thereby influencing both risk assessment and control strategies.
3. It can be hypothesized that particles from incomplete combustion have a noncancer health risk potential.

Major Unknowns:

1. There is evidence that there is a cancer potential associated with exposure to particulate matter, but the dimensions of that potential are not known.
2. The quantitative relationship between exposure to PM and delivered dose to man is relatively unknown for the indoor air situation. There is insufficient knowledge of personal activity patterns indoors, especially exercise levels, to permit application of existing and future pulmonary dosimetry models to evaluate the relationship between exposure and dose. The dosimetry, pharmacokinetics, and bioavailability of indoor organics adsorbed into particles is not known. These gaps in the data base prohibit even semiquantitative application of models relating monitored exposure to dose, dose being the factor that is critical to health effects. For example, ten people within an identical indoor environment, receiving identical exposures, could receive very different doses and hence experience different degrees of health risk, depending on whether these people had different activity patterns, different baseline pulmonary functions (i.e., males versus females, normals versus those with existing pulmonary disease), or different stages of lung development (i.e., child versus adult).
3. The noncancer health effects of indoor combustion particles, except for ETS, are almost totally unknown. Even if indoor PM was better characterized chemically, these effects would still be unknown, given the paucity of the general inhalation toxicology data base and the need to address PM in context of the

mixtures they are in. Given what is known, this huge gap in the data base is cause for concern.

2.3.9 Mitigation and Control Options for Combustion Sources

Indoor air controls that have application to combustion sources can be grouped in five general categories (Table 2-24).

TABLE 2-24. METHODS OF IAQ CONTROL FOR COMBUSTION SOURCES

Control Method	Examples
General Ventilation	Infiltration of outdoor air, whole building ventilation with outdoor air by mechanical equipment
Local Ventilation	Exhaust fan near a source (kitchen fan, range hood)
Air Cleaning	Filtration or electrostatic devices for particle removal in central air handling systems, absorption devices for gaseous pollutant removals
Source Modification	Proper tuning and operation of combustion appliances
Source Removal	Substitution of alternative nonpolluting source of heat
Building Design and Operation	Detachment of parking facilities, relocation of air intakes, maintenance of ventilation systems

General ventilation provides a multiple point source control by dilution with outdoor air. This type of air exchange can also introduce outdoor air pollutants into the occupied space. Therefore, general ventilation is only applicable when outdoor air has lower pollutant concentrations or if the air is cleaned prior to introduction. General ventilation can also make air quality in some areas of a building worse through the distribution of a pollutant. The energy requirements associated with whole-building ventilation make it impractical to increase the air change per hour (ach) to greater than 1 through mechanical means.

Avoiding unusually low ach is equally important for maintaining acceptable indoor air quality because indoor concentrations of contaminants appear to increase rapidly as ach decreases.

Local ventilation can be regarded as a source control measure for combustion sources because it effectively reduces pollutant source strength. Local exhausts have been demonstrated to minimize transport of pollutants from a source to an occupied space. Local ventilation can maintain low indoor pollutant concentrations for a concentrated source with less exhausted air and consequently less cost than general dilution ventilation. Local ventilation may have particular application to episodic operation such as occurs during the reloading of a wood burning stove.

Few data are available on the effectiveness of local exhaust from specific rooms. Evidence indicates that local exhaust can reduce transport of tracer gases to an adjoining room even when the doors between the rooms remain open, however, significant air flow rates are required. The practicality of exhausting a residence from an individual room has not been demonstrated.

Local exhausts such as range hoods are an important reduction strategy for NO_2. Range hoods have been demonstrated to reduce gas stove NO_x levels in kitchens by 30 percent at a flow rate of 93 l/s. The use of a range hood is demonstrated to be highly effective and to have near a linear relationship between range hood efficiency and flow rate. Such use should also effectively reduce concentrations of cooking aerosols.

Revza (1984) utilized sodium hexafluoride (SF6) as a surrogate pollutant source to evaluate the efficiency of range hoods and window fans operated as a local exhaust in a two-room test space (ach <0.1/hr). Using a range hood and both heated and unheated tracer gases he compared the concentrations of SF6 measured at multiple points within the space to those predicted by a simple two-room mass balance model. Revza found ventilation efficiency to be roughly linear over flow rates of 10.3 to 60 l/s for the heated tracer. The highest efficiency was 77 percent with heated tracer gas. Revza inferred that a flow rate approaching 75 to 100 l/s would produce 100 percent removal of the unheated tracer. With unheated tracer gas, the effectiveness of the range hood was found to be highly dependent on the ambient temperature. The variable buoyancy of the tracer made the calculation of an efficiency impossible (Revza, 1984).

Window fans were tested with the source in each of the two rooms and the fan remaining fixed. The fan was exhausting at a rate varying from 10.3 to 45.2 l/s. When the source was in the same room as the fan, the SF6 concentrations were found not to agree with the predictive model; when the source was in another room, fairly good agreement with the model was obtained. In neither case did the source differ by more than 50 percent from the predictive model (Revza, 1984).

Although use of air cleaners is an established and growing practice for IA control it does not appear to be directly applicable to inorganic combustion gases. There are two basic modes for applying air cleaners to indoor air. The first is to interpose the air cleaner between the source and the indoor environment, e.g., a recirculating range hood. The second is to treat the indoor air after the contaminant has been dispersed, e.g., central heating, ventilation, air conditioning (HVAC), or free-standing air cleaners. These technologies have potential application for removal of RSP and unburned hydrocarbons (UNBH), however, their efficiency and capacity for removal of specific pollutants have not been evaluated in indoor situations.

Residential furnace filters and panel filters such as those used by some portable air cleaners are not effective for particle removal. Electrostatic precipitators are demonstrated to be 50 to 90 percent efficient and extended surface area filters may remove 70 to 99 percent of the particles (Fisk et al., 1985).

The source modification control option for combustion gases has not been studied fully, even though some rather detailed work exists for the control of NO_x through modification of gas appliances. Modification can include replacement of standing pilot lights with other ignition devices such as electronic ignitions.

Zawacki et al. (1984) investigated simple methods of reducing NO_x emissions through modification in typical unvented gas space heaters. They reported that a significant reduction in NO_x could be obtained using appropriate burner inserts. This approach was not suitable for reduction of NO_2 (Zawacki et al., 1984).

Burner modifications, such as the use of wire mesh in the flame to conduct heat away from the hottest portion of the flame to reduce NO_2 formation, must be done in a way not to reduce the flame temperature. One mesh configuration has been shown to reduce NO_2 emissions by 70 percent with the concurrent 7500 percent increase of CO (Fisk et al., 1985). A double cylinder configuration was reported to reduce NO_x by approximately 62 percent while allowing CO burnout to proceed to completion; steel ring inserts in a range top burner demonstrated a 26 percent reduction in NO_2 without a dramatic increase in CO (Fisk et al., 1985). The basic strategy of burner inserts has been adopted recently in a modification introduced by the gas industry to meet the new California Standards for controlling NO_2 emissions from furnaces and water heaters (Fisk et al., 1985).

Source removal or source exclusion has application for the control of indoor air contamination arising from combustion sources. For example, the temperature for the formation of NO_2 which is produced in kerosene combustion appliances and in gas flames is not produced in appliances using electrical resistance elements. CO is also eliminated through the replacement of combustion sources with an appropriate electrical counterpart. SO_2 is produced by combustion of a fuel with a high sulfur content. It can be controlled through source elimination or fuel substitution.

Although not a source control, appropriate building design and operation as a control option is the goal of many building professionals. Design may include the location of specific subunits and systems as well as the selection of building materials. Because CO is a major exhaust pollutant from internal combustion engines, the isolation of garages and loading docks from occupied building is an acceptable design strategy. Where this cannot be accomplished it appears essential to monitor the leakage paths into the occupied areas for CO. The location of the air intakes and the location and condition of the ducting system are also crucial. Most air pollutants appear to increase in concentration from the air intake along the path of the duct work of the ventilation system (Berglund et al., 1982).

The deliberate use of a pollutant's reactivity also has potential as a design strategy. However, before reactivity can be considered as a control strategy it is essential to understand the mechanism of a pollutant's generation and decay. Indoor air pollutant concentration is a function of pollutant source strength, air transport and diffusion, and the chemical reactions occurring within and on the various surfaces of a building. The chemical reactivity and the internal air flow are both affected by temperature, and reactivity and may be affected by humidity as well (GEOMET Technologies, Inc., 1976).

The use of reactivity to control NO_2 has been proposed by Fisk and coworkers (1985), who reported a removal rate by reactivity of 0.16 to 1.29 per hour for NO_2. Sulfur dioxide is also a highly reactive compound and may be differentially adsorbed by interior walls, ceilings, and floor coverings. Consequently, design changes which increase air circulation or inclusion of specific surface material may be helpful in the removal of SO_2 (Fisk et al., 1985).

Proper operation and maintenance are also important control options. In those occupied spaces affected by leaking combustion products, the replacement of cracked heat exchangers in furnaces, or improvements to the venting system can provide control.

There are basically two strategies for RSP control. The first is source exclusion or reduction; the second is the removal of RSP from the air. Reduction in the emissions from wood stoves and fireplaces can be accomplished through structural changes such as wood stove inserts in a fireplace, or by replacing fireplace screens with tempered glass doors, or by operational changes, such as mode of loading, amount of air supplied, or type and dryness of wood burned.

2.3.9.1 Major Knowns and Unknowns. The general absence of data and the absence of reliable particulate emission factors from the indoor sources makes it impossible to establish a relative ranking between reported sources and their contribution to indoor air pollution. In general, the absence of standardized test procedures, including sampling and analysis methods, and lack of uniformity in test conditions precludes the use of much of the existing data for the predictive modeling which facilitates risk assessment calculations.

Major sources of uncertainty related to indoor air combustion particulates are the source and emission rates of particulates, the characteristic size distribution, the presence and identity of absorbed chemical compounds, and the applicability of a given control option to a specific pollutant source.

Indoor air source characterization unknowns related to particles emitted from combustion sources include the following:

- characterization of various sources with particular emphasis on size and mass distribution of particles produced by unvented sources
- expanded characterization of particle-bound organics from combustion devices
- survey of indoor combustion sources to gather statistics on age, condition, and operational parameters of combustors
- investigation of the factors affecting leakage rates of particles from vented combustion sources
- studies of sink rates of particles on indoor materials

2.4 ENVIRONMENTAL TOBACCO SMOKE

2.4.1 Introduction

Tobacco smoke is a major contributor of particulate and organic matter in the indoor environment (Samfield, 1985; National Research Council, 1981b; Repace, 1982). The health effects of smoking on smokers has been extensively studied, but the effect of passive smoking on nonsmokers has received far less attention (Taylor et al., 1978; Surgeon General of the U.S., 1986; National Research Council, 1986a).

Smoking contributes enormously to RPM in buildings. The following are some results from the Harvard Six Cities Study (Spengler et al., 1981; Samfield, 1985) which indicate that smoking can triple I/O ratios of RPM.

Location	Mean Concentration $\mu g/m^3$	I/O Ratio
Outdoor	21.1	-
Indoor - no smoking	23.4	1.16
Indoor - 1 smoker	36.5	1.73
Indoor - 2 smokers	70.4	3.34

Nonsmokers inhale environmental tobacco smoke, which is the combination of sidestream smoke (SS) released into the air from the cigarette's burning end, and mainstream smoke (MS), exhaled by the active smoker. Because tobacco smoking releases myriad chemical species into the air, ETS is an extremely complex mixture that changes as it ages. The exposures to passive and active smoking differ qualitatively and quantitatively (U.S. Department of Health and Human Services, 1984). SS has higher concentrations of some toxic and carcinogenic substances than MS; however, dilution by room air markedly reduces the concentrations inhaled by the passive smoker in comparison with those inhaled by the active smoker. Individual components of tobacco smoke can be measured in indoor air, but no single component has been shown to accurately measure the disease-causing potential of ETS.

Klus and Kuhn (1982) actually measured the mainstream and sidestream emissions of various compounds in cigarette smoke. Some of their results are given in Table 2-25. Note that ammonia appears in the sidestream at a ratio of 10-24:1 to that in the mainstream. This may explain the somewhat more basic character of SS to MS.

TABLE 2-25. SOME SIDESTREAM TO MAINSTREAM (SS/MS) RATIOS DETERMINED FOR VARIOUS ELEMENTS AND COMPOUNDS IN CIGARETTE SMOKE

Substance	SS/MS
Nicotine	2.6 to 3.3
Carbon monoxide	2.5 to 4.7
Carbon dioxide	8.9 to 11.3
Propanal	approximately 2.5
Formaldehyde	approximately 51
Acrolein	approximately 12
Hydrogen cyanide	0.056 to 0.37
Acrolein	3.41 to 5.30
Hydrogen cyanide	73 to 170
Acetonitrile	29.7
Ammonia	10 to 24
Aniline	19.1 to 100
Pyridine	10 to 24
N-nitrosodimethylamine	19.1 to 100
N'-nitrosonornicotine	0.48 to 2.7
N'-nitrosoanatabine	0.31 to 0.95
4-(N'-methyl-N'-nitrosoamino)-1(3-pyridyl)-1-butanone	1.27 to 3.7
Toluol	5.6 to 83
Benzo(a)pyrene	2.1
Quinoline	10.8
Cadmium	3.58 to 7.2
Nickel	0.19 to 31.0
Zinc	0.2 to 6.66

Repace (1984) and co-workers have conducted extensive field studies and measured the CO and particulate phase emissions in rooms where cigarette smokers were present. One of their conclusions was that there is good evidence for treating the emissions of all cigarettes alike. He hypothesized a tar yield of 32.5 mg/cigarette. Repace and Lowry (1985) estimated that the "typical" U.S. nonsmoker is exposed to 1.4 mg of tobacco tar per day; the range for U.S. nonsmokers is given as 0 to 14 mg/day (Samfield, 1985).

White and Froeb (1980) concluded that chronic exposure to tobacco smoke in the workplace is equivalent to smoking 1 to 10 cigarettes per day. Several epidemiological studies purport to show that nonsmoking spouses of smokers are at a high risk than nonsmokers married to nonsmokers (Hirayama, 1981; Garfinkel, 1981).

2.4.2 Source Characterization

As the sidstream smoke and exhaled mainstream smoke, together with other gases and particles emitted from burning tobacco, are diluted and circulated in indoor air, the chemical and physical character of the constituents are thought to change substantially from either MS or SS, based upon those few constituents which have been studied in ETS. The median diameter of particles in ETS is smaller than in undiluted SS (Wynder and Hoffmann, 1967). Nicotine, which is primarily associated with the particle phase of undiluted SS, is thought to redistribute between the particle and vapor phase as the SS dilutes, so that it is found almost exclusively in the vapor phase in ETS (Eudy et al., 1985, 1986; Hammond et al., 1987). More data are needed on the distribution of ETS components between the gas and particle phases since this factor is critical to the human dosimetry and retention of ETS components in the lung.

Most of the chemical characterization data available which is pertinent to ETS comes from studies that demonstrated that 300 to 400 of the more than 3800 constituents in MS have also been measured in SS (Wynder and Hoffman, 1967). Certain constituents are found in much higher concentrations in SS as compared to MS, including the carcinogenic volatile nitrosamines and the volatile pyridines. Since SS also has high concentrations of nitrogen dioxide, which is a nitrosating agent, chemical transformations are expected to occur between the point at which chemicals are emitted as SS and the point at which nonsmokers are exposed to ETS. Another change which occurs as SS is diluted to form ETS is that in the presence of radon, the short-lived radon daughters will attach to ETS particles and become more available for inhalation than in the absence of ETS. Bergman et al. (1986) have shown that indoor radon-daughter concentrations can more than double in the presence of tobacco smoke. It is critical, therefore, that research be conducted to determine the presence, concentration and phase distributions of toxic and carcinogenic constituents of ETS in indoor air spaces. Interactions with non-smoke toxic compounds need to be clarified. Special consideration needs to be given to the possibility of secondary reactions of amines in indoor air containing ETS that could result in the production of carcinogenic N-nitrosation and condensation products.

A critical link between source characterization and exposure assessment is source emission factors. Studies have been conducted to determine particle, tar, nicotine, and CO emission factors for SS. Rickert et al. (1984) showed that these SS emission factors were independent of MS emissions. These and other studies suggest that the SS emission factors for these components are similar across all brands of cigarettes. Unfortunately, most of these studies have been conducted using SS collection devices rather than chamber studies where ETS collection is performed. Studies are needed to determine the emission factors and surface removal rates for critical constituents of ETS. The emission rates for carcinogenic and toxic constituents especially need to be determined.

2.4.3 Exposure Assessment

Three approaches can be used to assess human exposure to ETS:

1. Exposure modeling using emission rates, dispersion models, and time-activity patterns
2. Monitoring exposure concentrations by using either personal monitoring or microenvironmental measurements combined with time-activity patterns
3. Human exposure monitoring using biological markers of exposure

All three approaches are being used to address the important question of human exposure assessment to ETS. Unfortunately, the most imprecise, yet simplest method, that of employing a simple questionnaire (e.g., Do you live with, or work with, or have regular contact with persons who are smokers?), has been the most widely used method of estimating human exposure to ETS. This method has been used to classify individuals into broad categories of exposure while recognizing the problems associated with misclassification due to errors in reporting current smoking habits, neglecting certain exposures, and the reporting of exsmokers as nonsmokers.

2.4.3.1 Exposure Modeling. A model for exposure of nonsmoking adults in the U.S. to ETS particles has been recently developed by Repace and Lowrey (1985) and is expanded upon by Repace (in press). This model is dependent upon particle emission factors, time-dependent growth and decay of ETS concentrations in indoor air spaces, and ventilation and mixing of ETS in room air, to develop a general equilibrium model and equation for ETS. This model predicts that the exposure of U.S. nonsmokers ranges from 0 to 14 mg of cigarette tar per day, depending upon the nonsmokers's lifestyle. The average population exposure for adults of working age, averaging over the work and home microenvironments, is about 1.43 mg/day (Repace and Lowrey, 1983) with an 86 percent exposure probability.

Exposure modeling methods for ETS have recently been reviewed by the National Research Council (1986a). The modeling efforts to date have concentrated on modeling the respirable particles (RSP) (particles <2.5 microns) emitted from ETS in indoor environments. These models generally use a simplified form of the mass-balance equation. The models are generally single-compartment models that assume steady-state or equilibrium conditions to estimate RSP concentrations. The input parameters are the RSP-emission rate for ETS, number of cigarettes consumed, ventilation or infiltration rates, removal rates by surfaces, air mixing in the environment, and volume of the space. The variability of one or more of the input parameters can make a difference of as much as an order of magnitude in the estimated RSP concentration (National Research Council, 1986a). To the extent that the equilibrium assumptions do not hold, for example if the source is intermittent rather than continuous, then additional variability in the exposure assessment is introduced.

The limited field tests of these general equilibrium models have predicted RSP concentrations reasonably well over a wide range of input values for the equations (National Research Council, 1986a). Using this approach, the predicted RSP exposures due to ETS are highly variable, but always consistently predict substantial increases in human exposure when smoking occurs in the indoor space being modeled. The National Research Council (1986a) recommends that when field measurement studies of ETS constituents are conducted, data on model input parameters (e.g., smoking rates, room volume, air exchange rate, etc.) should be collected so that field evaluations of the equilibrium model can be made. More information is also needed on current and past distributions of the input parameters for the mass-balance models of RSP concentrations for a range of microenvironments in which individuals spend most of their time.

2.4.3.2 Monitoring Exposure. Measurement and monitoring of ETS are hampered by the fact ETS is a very complex mixture of organic and inorganic gases and particles. All of the thousands of ETS constituents cannot be monitored on a routine basis, therefore a marker or tracer for quantifying ETS concentrations indoors and personal exposure is needed. The National Research Council (1986a) reviews the tracers that have been used for ETS and suggest that a suitable tracer should:

- be unique or nearly unique to tobacco smoke;
- be present in sufficient quantities to be detected at low smoking rates;
- have similar emission rates for a variety of tobacco products; and
- have a fairly consistent ratio to the constituents of interest (e.g., the carcinogenic constituents) over a range of environmental conditions and for a variety of tobacco products.

While several constituents have been used as tracers of ETS, no single measure has met all the criteria listed by the NAS, nor has any one of these measures been recognized as representing ETS exposure. The constituents reviewed by the National Research Council (1986a) which have been examined as tracers include: CO, RSP, nicotine, aromatic hydrocarbons, tobacco-specific nitrosamines, nitrogen oxides, acrolein, acetone, and polonium-210. The NAS report repeatedly recommends that research efforts need to be directed toward identifying a tracer contaminant for ETS that meets the criteria listed above. At present, RSP is the most widely used as a general measure of ETS exposure indoors. Smoking, however, is not the only source of RSP in indoor air. Nicotine has many of the characteristics necessary to serve as a tracer for ETS. The major problems with nicotine as a tracer are: (1) the ratio of nicotine to other ETS constituents for a variety of tobacco products is not known, (2) the reactivity rate of nicotine relative to other ETS constituents is not known, (3) particulate- or vapor-phase nicotine may be re-emitted once deposited on surfaces, and (4) until very recently, efficient sampling methods for total nicotine were not available.

The most extensive indoor air monitoring data base on a constituent associated with ETS is that for RSP. In addition to microenvironmental measurements of indoor air spaces, personal monitoring methods are available for RSP. Several studies have recently utilized personal monitors of either RSP or nicotine to determine total human exposure to ETS (Muramatsu et al., 1984; Schenker et al., 1984; Sexton et al., 1984; Spengler et al., 1985, Hammond et al., in press). Spengler et al. (1981) has conducted an extensive study of exposure to RSP indoors and outdoors, and more recently (Spengler et al., 1985) using personal monitors. These studies demonstrate the dominant role of ETS in increasing RSP levels indoors and in personal exposures to RSP.

2.4.3.3 Biological Markers. Exposure to ETS depends upon many factors. The National Research Council (1986a) concluded that an optimal assessment of exposure should be done by analysis of the physiological fluids of exposed individuals rather than just relying upon the indoor air assessment. The methods which have been used to date are reviewed by the National Research Council (1986a) and the Surgeon General (1986). These methods include measurements in physiological fluids of: thiocyanate, carboxyhemoglobin, nicotine and its metabolite cotinine, hydroxyproline, N-nitrosoproline, aromatic amines, and urinary mutagens.

Nicotine and cotinine are currently the best available markers of human exposure to ETS. Both nicotine and cotinine in biological fluids are highly specific to tobacco smoke exposure. Cotinine measurements offer several advantages over nicotine, and particularly because of its long half-life in adult nonsmokers, it has become the current method of choice for assessing human exposure to ETS. In order to use this biological marker more effectively, improved and standardized methods for measuring cotinine are needed, and studies quantifying the relationship between exposure, dose, and clearance rate for nonsmokers of diverse age, sex, and race are needed. Current studies being conducted by EPA suggest that the clearance rate for cotinine in infants is much faster than in adults.

Nicotine in ETS appears to be primarily in the vapor phase and may not be directly related to the carcinogenic potential of ETS. The National Research

Council (1986a) recommends that indicators that are related to the carcinogenic risk are needed. Sensitive dosimetry methods for tobacco-specific compounds are urgently needed. They suggest that new methods for immunoassays and postlabelling be used in the development of dosimetry studies in nonsmokers exposed to ETS. Protein and DNA adducts are recommended as possible exposure and dosimetry measures that could be effectively used in both exposure and epidemiologic studies.

2.4.4 Health Effects

2.4.4.1 Introduction. The health effects of environmental tobacco smoke (ETS) or passive smoking have recently been thoroughly reviewed by the World Health Organization (International Agency for Research on Cancer, 1986), National Research Council of the National Academy of Sciences (National Research Council, 1986a) and the U.S. Department of Health and Human Services (Surgeon General of the U.S., 1986). Effects can be divided into acute irritating and immune effects, respiratory effects, cancer, and cardiovascular effects, among others.

2.4.4.2 Acute Irritating and Immune Effects. The most common acute effects associated with exposure to ETS are eye, nose and throat irritation, and objections to the smell of tobacco smoke. Particle filtration causes little decline in irritation or odor, which suggests that these effects may be caused by gas-phase rather than particle-phase constituents of ETS (National Research Council, 1986a).

Eye-blink rate as a measure of irritation correlates positively with ETS concentration and is likewise correlated with sensory irritation such as burning eyes or nasal mucosa (Weber-Tschopp et al., 1976; Weber, 1984).

Cigarette smoke contains immunogens, which are substances that activate the immune system. Approximately one-half of allergy-prone individuals react to tobacco extracts or smoke extracts in skin tests, but the specific immunogenic compounds have not been isolated from these extracts. Self-reported complaints of sensitivity to smoke do not correlate well with skin-sensitivity tests (Lehrer et al., 1984).

2.4.4.3 Respiratory Effects. The evidence for health effects from involuntary exposure to tobacco smoke is strong for children. Such exposure is currently widespread, and because of its high prevalence even small effects on morbidity and mortality have important public health implications.

Epidemiological investigations have linked passive smoking in children to increased occurrence of lower respiratory tract illness (presumably of infectious etiology) during infancy (Table 2-26), to increased prevalence of respiratory symptoms, and to reduction of lung function. These studies indicate a significantly increased frequency of bronchitis and pneumonia during the first year of life in children with smoking parents. Although outcome measures varied somewhat among the studies, the relative risks associated with passive smoking were similar, and dose-response relationships between infant illness and extent of parental smoking were demonstrable. An effect of passive smoking was not readily identified after the first year of life. Retrospective collection of information from the parents of older children confirms excessive lower respiratory illness experienced by infants whose parents smoke (Schenker et al., 1983; Ware et al., 1984).

For respiratory symptoms in children, the effect of passive smoking is not as large as for lower respiratory illness (Table 2-27). However, data from numerous cross-sectional studies demonstrate a greater frequency of the most common respiratory symptoms--cough, phlegm, and wheeze--in the children of smokers (U.S. Department of Health and Human Services, 1984). In these studies the subjects have generally been schoolchildren. Recent results from several large studies, the Harvard Air Pollution Health Study (Ware et al., 1984) and an English study reported by Charlton (1984) provide convincing evidence that passive smoking increases the occurrence of cough and phlegm in the children of smokers, although earlier data from smaller studies had been ambiguous (Table 2-26). The evidence also indicates an excess of chronic wheeze associated with longitudinal investigations have provided the previously missing evidence of the consequences of passive smoking during lung growth and development. Tager et al. (1979) reported that the FEF_{25-75} declined with the number of smoking parents in the household, based on cross-sectional data from children in East Boston, Massachusetts.

In 1983, these investigators provided follow-up results for these children over a seven-year period (Tager et al., 1983). Using a multivariate statistical analysis, they showed that both maternal smoking and active smoking by the child reduced the growth rate of the FEV_1. Longitudinal data from the passive smoking (Table 2-26), although passive smoking has not been significantly associated with wheezing in all studies (Schenker et al., 1983; Leeder et al., 1976; Tashkin, 1984). Evidence for association of passive smoking with

TABLE 2-26. PREVALENCE OF RESPIRATORY SYMPTOMS IN SELECTED INVESTIGATIONS OF CHILDREN, BY NUMBER OF SMOKING PARENTS*

Subjects	Respiratory System	Prevalence (per 100) by Number of Smoking Parents		
		0	1	2
2426 children, aged 6 to 14, England (Colley, 1974).	Chronic cough	15.6	17.2	22.2
3105 nonsmoking children, aged 12 to 13, England (Bland et al., 1978).	Cough during the day or at night	16.4	19.0	23.5
650 children, aged 5 to 9, Massachusetts (Weiss et al., 1980).	Chronic cough and phlegm	1.7	2.7	3.4
	Persistent wheeze	1.8	6.8	11.8
4071 children, aged 5 to 14, Pennsylvania (Schenker et al., 1983).	Chronic cough	6.3	7.0	8.3
	Chronic cough	4.1	4.8	4.0
	Persistent wheeze	7.2	7.7	5.4
8528 children, aged 5 to 9, six U.S. cities (Ware et al., 1984).	Chronic cough	7.7	8.4	10.6
	Persistent wheeze	9.9	11.0	13.1
1733 nonsmoking children aged 8 to 10, England (Charlton, 1984)	Frequent cough (boys)	35	42	48
	Frequent cough (girls)	32	40	52

*Abstracted from Table 4, U.S. Department of Health and Human Services (1984) and from Charlton (1984).

the development of childhood asthma is conflicting (Gortmaker et al., 1982; Burchfiel, 1984; Leeder et al., 1976; Horwood et al., 1985; Schenker et al., 1983; Tashkin et al., 1984).

Substantial data are now available on the effects of parental smoking on children's lung function. The Surgeon General's 1984 report on smoking and health (U.S. Department of Health and Human Services, 1984), based on cross-sectional observations, concluded that passive smoking had a small but measurable effect on the lung function of children. However, the long-term consequences of these changes were considered to be unknown. More recent data

TABLE 2-27. EPIDEMIOLOGICAL STUDIES OF EARLY CHILDHOOD RESPIRATORY ILLNESSES AND PASSIVE SMOKING

Study Population	Study Design	Effect of Passive Smoking	Comments
10,672 births in Israel, 1967-1968 (Harlap, 1974)	Antenatal maternal smoking history, monitoring of admissions during first year of life	Significant increase in hospitalization for pneumonia and bronchitis, RR* = 1.4	Dose-response relationship present. Maternal smoking only
2205 births in England, 1963-1965 (Leeder et al., 1976)	Prospective cohort with annual questionnaire	Significant increase in bronchitis or pneumonia in first year of life, RR = 1.7 if one parent smoked, RR = 2.6 if both smoked	Sex of smoking parent not examined
12,068 births in Finland, 1966 (Rantakallio, 1978)	Prospective cohort with follow-up of hospitalizations, physician visits, and mortality	Significant increase of hospitalization for respiratory diseases during first 5 years, RR = 1.7	Effect largest during first year. Maternal smoking only and measured during pregnancy
1265 births in New Zealand, 1977 (Fergusson et al., 1985)	Prospective cohort with diaries, physician and hospital record review	Significant increase in bronchitis or pneumonia during the first year of life, RR = 2.0, if mother smoked	No effect of paternal smoking. Effect of maternal smoking equivocal in second year, absent in third. Dose-response present in first
130 children with respiratory virus infection in infancy, England (Pullan and Hey, 1982)	Case-control with 111 controls, performed 10 years after index illness	Significant effect of maternal smoking at time of illness, RR = 3.2	Effect of paternal smoking also present
1058 births in China, 1981 (Chen et al., 1986)	Prospective cohort with illnesses ascertained at age 18 months by a questionnaire	Significant effect of smoke exposure at home, RR = 1.9 if >10 cigarettes per day consumed by family	None of the mothers smoked

*RR = relative risk. Calculated from published data if not provided by the authors.

from three longitudinal investigations have provided the previously missing evidence of the consequences of passive smoking during lung growth and development. Tager et al. (1979) reported that the FEF_{25-75} declined with the number of smoking parents in the household, based on cross-sectional data from children in East Boston, Massachusetts.

In 1983, these investigators provided follow-up results for these children over a seven-year period (Tager et al., 1983). Using a multivariate statistical analysis, they showed that both maternal smoking and active smoking by the child reduced the growth rate of the FEV_1. Longitudinal data from the Harvard Air Pollution Health Study (Ware et al., 1984) also showed reduced growth of the FEV_1 in children whose mothers smoked cigarettes (Berkey et al., 1986). In children aged six to ten years, a statistical model estimated that FEV_1 growth rate is reduced by 0.17 percent per pack of cigarettes smoked daily by the mother. Burchfiel (1984) examined the effects of parental smoking on 15-year lung function change of subjects in the Tecumseh study, first examined at ages 10 through 19 years. Among subjects who remained nonsmokers across the follow-up period, parental smoking reduced the growth of the FEV_1, the FVC, and the $Vmax_{50}$ in males but had no effect in females.

The health effects of passive smoking on adults have not been as comprehensively examined and therefore remain controversial. Only a few cross-sectional investigations provide information on the association between respiratory symptoms in nonsmokers and passive smoking, and their results do not provide consistent evidence of an effect of passive smoking (Lebowitz, 1976; Schilling et al., 1977; Comstock et al., 1981; Schenker et al., 1982).

With regard to pulmonary function in adults, exposure to passive smoking has been associated with reduction of the FEF_{25-75} in two cross-sectional investigations. White and Froeb (1980) compared spirometric test results in middle-aged nonsmokers with at least 20 years of passive smoking exposure in the workplace to the results in a control group who did not have similar exposure. The mean FEF_{25-75} of the exposed group was significantly reduced, by 15 percent of the predicted value in women, and 13 percent in men. A recent population-based French investigation examined the effect of marriage to a smoker in 849 male and 826 female nonsmokers (Kauffmann et al., 1983). Above age 40 years, the FEF_{25-75} was reduced in nonsmoking men and women with a smoking spouse. The results of an investigation of 163 nonsmoking women in the Netherlands also suggested adverse effects of tobacco smoke exposure in the home (Remijn et al., 1985; Brunekreef et al., 1985).

Other studies have not indicated chronic effects of passive tobacco smoke exposure on adult nonsmokers. In two cross-sectional studies, marriage to a smoker was not significantly associated with reduction of ventilatory function (Schilling et al., 1977; Comstock et al., 1981). A case-control study of 20- to 39-year-old nonsmoking women in the Tecumseh Community Health Study cohort also did not show an effect of marriage to a smoker on lung function level (Burchfiel, 1984). Kentner et al. (1984) examined the effects of passive and active smoking in 1351 German white collar workers. Self-reported exposure to ETS at home and at work was not associated with reduction of lung function, as assessed by spirometry.

Neither epidemiological nor experimental studies have established the importance of ETS in exacerbating asthma in adults. Of three studies involving experimental exposure of adult asthmatics to tobacco smoke (Shephard et al., 1979; Dahms, 1981; Wiedemann et al., 1986), only one showed a definite adverse effect (Dahms, 1981). In a population sample in Tucson, AZ, Lebowitz et al. (1984) examined the relationship between passive smoking and daily symptom occurrence and daily level of peak flow. Peak flow rates in the asthmatic participants were not affected by ETS exposure, although effects on symptom status were reported.

In 1981, reports were published from Japan (Hirayama, 1981) and Greece (Trichopoulos et al., 1981) that showed increased lung cancer risk in nonsmoking women married to cigarette smokers. Subsequently, involuntary exposure to tobacco smoke has been examined as a risk factor for lung cancer in studies conducted throughout the world (Table 2-28). While not all of the studies have shown significantly increased risk associated with exposure, the weight of the epidemiological evidence supports a conclusion that involuntary exposure to tobacco smoke is a risk factor for lung cancer. This conclusion can be further supported on a biological basis. SS contains many of the same carcinogens present in MS; tobacco smoke materials are absorbed during involuntary exposure; and respiratory carcinogenesis appears to take place without any threshold of exposure. These biological considerations underlie the recent conclusion by the International Agency for Research on Cancer of the World Health Organization (1986) that "passive smoking gives rise to some risk of cancer."

All of the epidemiological data discussed here provide evidence of adverse effects in children and adults exposed to ETS. In all of the studies exposure

TABLE 2-28. COHORT AND CASE-CONTROL STUDIES OF PASSIVE EXPOSURE TO TOBACCO SMOKE AND LUNG CANCER

Study	Findings	Comment
Prospective cohort study in Japan of 91,540 nonsmoking females, 1966-1981 (Hirayama, 1984)	Age-occupation adjusted SMRs, by husband smoking: Nonsmokers - 1.00 Exsmokers - 1.36 1-19/day - 1.45 >20/day - 1.91	Trend statistically significant; All histologies
Case-control study in Greece of 40 nonsmoking female cases, 149 controls, 1978-1980 (Trichopoulos et al., 1981)	Odds ratios by husband smoking: Nonsmokers - 1.0 Exsmokers - 1.8 Current smokers <20/day - 2.4 >21/day - 3.4	Trend statistically significant; Histologies other than adenocarcinoma and bronchioloalveolar carcinoma
Prospective cohort study in the U.S. of 176,139 nonsmoking females, 1960-1972 (Garfinkel, 1981)	Age-adjusted SMRs, by husband smoking: Nonsmokers - 1.00 Current smokers <20/day - 1.27 >20/day - 1.10	All histologies; Effect of husband smoking not significant
Case-control study in Hong-Kong of 84 female cases and 139 controls, 1976-1977 (Chan et al., 1979; Chan and Fung, 1982)	Crude odds ratio of 0.75 associated with smoking spouse.	All histologies. Two reports are inconsistent on the exposure variable
Case-control study in the US with 22 female and 8 male nonsmoking cases, 133 female and 180 male controls (Correa et al., 1983)	Odds ratios by spouse smoking: Nonsmokers - 1.00 <40 pack years - 1.48 >41 pack years - 3.11	Significant increase for >41 pack years. Bronchioloalveolar carcinoma excluded
Case-control study in the U.S.A. 25 male and 53 nonsmoking female cases with matched controls, 1971-1980 (Kabat and Wynder, 1984)	Odds ratio not significantly increased for current exposure at home: Males - 1.26 Females - 0.92	All histologies. Findings negative for spouse smoking variable as well

(continued on following page)

TABLE 2-28. (continued)

Study	Findings	Comment
Prospective cohort study in Scotland of 8128 males and females, 1972-1982 (Gillis et al., 1984)	Age-adjusted mortality ratios for domestic exposure: Males - 3.25 Females - 1.00	Preliminary, small numbers of cases
Case-control study in Hong Kong with 88 nonsmoking female cases, 1981-1982 (Koo et al., 1984)	Odds ratio of 1.24 ($p > 0.40$) for combined home and workplace exposure. No association with cumulative hours of exposure	All histologies
Case-control study in the U.S. with 31 nonsmoking and 189 smoking female cases (Wu et al., 1985)	No significant effects of exposure from parents, spouse, or workplace in smokers and nonsmokers	Adenocarcinoma and squamous cell carcinoma only
Case-control study in the U.S. with 134 nonsmoking female cases (Garfinkel et al., 1985)	Nonsignificant odds ratio of 1.22 if husband smoked. Significantly increased odds ratio of 2.11 if husband smoked 20 or more cigarettes daily at home. Significant trend with number of cigarettes smoked at home by the husband	All histologies; Careful exclusion of smokers from the case group
Case-control study in Japan with 19 male and 94 female nonsmoking cases, and 110 male and 270 female nonsmoking controls (Akiba et al., 1986)	For females, odds ratio of 1.5 if husband smoked; for males, odds ratio of 1.8 if wife smoked	Clinical or radiological diagnosis for 43%. All histologies
Case-control study in Louisiana, Texas, and New Jersey with 99 nonsmoking cases and 736 controls (Dalager et al., 1986)	Adjusted odds ratio for marriage to a smoking spouse was 1.5	Nearly 100% histological confirmation. All histologies

SMR = standard mortality ratio

has been assessed qualitatively rather than by measurements of personal exposure or smoke concentrations in rooms. Information of quantitative dose-response relationships for these effects is lacking.

2.4.4.4 Lung Cancer and Other Cancers. Smoking remains the largest single preventable cause of death and disability for the U.S. population. Recently the WHO/IARC issued a comprehensive monograph (International Agency for Research on Cancer, 1986) on the carcinogenic risk of tobacco smoke to humans, which concludes that cigarette smoking is a major cause of cancer and is most strongly associated with cancers of the lung and respiratory tract. Smoking also causes cancers at other sites, including the pancreas and urinary bladder.

The National Research Council (1986a) and the Surgeon General of the U.S. (1986) released reports in December of 1986 on ETS which included extensive reviews of studies examining cancer risk from passive smoking or involuntary exposure to tobacco smoke. The summary that follows is taken from these two reports.

Exposure to ETS has been examined as a risk factor for lung cancer in nonsmokers in numerous recent epidemiologic studies. These studies have compared the risks for subjects exposed to ETS with the risks for people not reported to be exposed. Because exposure to ETS is an almost universal experience in the more developed countries, these studies involve comparison of more exposed and less exposed people, rather than comparison of exposed and unexposed people. The studies are therefore inherently conservative in assessing the consequences of exposure to ETS. Interpretation of these studies must consider the extent to which populations with different ETS exposures have been identified, the gradient in ETS exposure from the lower exposure to the higher exposure groups, and the magnitude of the increased lung cancer risk that results from increased ETS exposure.

To date, questionnaires have been use to classify ETS exposure. Quantification of exposure by questionnaire, particularly lifetime exposure, is difficult and has not been validated. However, spousal and parental smoking status identify individuals with different levels of exposure to ETS. Therefore, investigation has focused on the children and nonsmoking spouses of smokers, groups for whom greater ETS exposure would be expected, and for whom increased nicotine absorption has been documented compared to the children and nonsmoking spouses of nonsmokers.

Most (11 of 13) epidemiologic studies reviewed that have examined the association between involuntary smoking and lung cancer have shown a positive association with exposure, and in 6 the association reached statistical significance (Surgeon General of the U.S., 1986; National Research Council, 1986a).

There were five additional studies that were excluded because of inadequacies either of documentation or of the study itself. The NRC report summarized the relative risk estimate from each of these studies and determined a summary estimate based on the combined studies.

This risk was 1.34 (95 percent confidence limits 1.18 to 1.53) overall (National Research Council, 1986a). For all women the relative risk was 1.32 (1.16 to 1.51); for men it was 1.62 (0.99 to 2.64). The wide confidence limits for men reflect the fact that most of the data were based on nonsmoking women rather than nonsmoking men. For studies conducted in the United States, the relative risk was 1.14 (0.92 to 1.40). Considering only the largest studies (those with expected number of lung cancer deaths of 20 or more), the relative risk estimate was 1.32 (1.15 to 1.52) (National Research Council, 1986a). The confidence limits on each of these estimates all include the overall summary estimate of 1.34. Given the difficulty in identifying groups with differing ETS exposures, the low-dose range of exposure examined, and the small numbers of subjects in some series, it is not surprising that some studies have found no association, and that in others the association did not reach a conventional level of statistical significance. The question is not whether cigarette smoke can cause lung cancer; that question has been answered unequivocally by examining the evidence for active smoking. The question is, can tobacco smoke at a lower dose and through a different mode of exposure cause lung cancer in nonsmokers? The answer must be sought in the coherence and trends of the epidemiologic evidence available on this low-dose exposure to a known human carcinogen. In general, those studies with larger population sizes, more carefully validated diagnosis of lung cancer, and more careful assessment of ETS exposure status have shown statistically significant associations. A number of these studies have demonstrated a dose-response relationship between the level of ETS exposure and lung cancer risk. By using data on nicotine absorption by the nonsmoker, the nonsmoker's risk of developing lung cancer observed in human epidemiologic studies can be compared with the level of risk expected from an extrapolation of the dose-response data for the active smoker. This extrapolation yields estimates of an expected lung cancer risk that approximate the observed lung cancer risk in epidemiologic studies of involuntary smoking.

The weight of evidence derived from epidemiologic studies shows an association between ETS exposure of nonsmokers and lung cancer that, taken as a whole, is unlikely to be due to chance or systematic bias. The observed estimate of increased risk is 34 percent, largely for spouses of smokers compared with spouses of nonsmokers. One must consider the alternative explanations that this excess either reflects bias inherent in most of the studies or that it represents a causal effect. Misclassification of smokers, exsmokers and nonsmokers may have contributed to the result to some extent. Computations of the effect of two sources of misclassification were presented. Computations taking into account the possible effects of misclassified exsmokers and the tendency for spouses to have similar smoking habits placed the best estimate of increased risk of lung cancer at about 25 percent in persons exposed to ETS, at a level typical of that experienced by nonsmokers married to smokers, compared with those married to nonsmokers. Another computation using information from cotinine levels observed in nonsmokers, and taking into account the effect of making comparisons with a reference population that is truly unexposed, leads to an estimated increased risk of about one-third when exposed spouses were compared with a truly unexposed population.

The data presented in the Surgeon General and NRC reports establish that a substantial number of the lung cancer deaths that occur among nonsmokers can be attributed to involuntary smoking. However, better data on the extent and variability of ETS exposure are needed to estimate the number of deaths with confidence.

2.4.4.5 Cardiovascular Disease and Other Effects. Tobacco smoking and cardiovascular disease are causally associated (U.S. Department of Health and Human Services, 1984), and the effects of smoking on exercise tolerance and blood pressure are well documented and reviewed elsewhere (U.S. Department of Health and Human Services, 1984). The constituents of tobacco smoke thought to be associated with cardiovascular disease are CO and nicotine.

The effects of ETS on cardiovascular disease have recently been reviewed by the National Research Council (1986a) and the Surgeon General of the U.S. (1986). A summary from the Surgeon General's report follows.

The relationship between cardiovascular disease and ETS has been examined in one case-control study and three prospective studies. In the case-control study by Lee and colleagues (1986), ischemic heart disease cases and controls did not show a statistically significant difference in their exposure to ETS based on the smoking habits of spouses or on an index accounting for exposure at home, at work, and during travel and leisure. In a Japanese cohort study, Hirayama (1984, 1985) reported an elevated risk for ischemic heart disease (N = 494) in nonsmoking women married to smokers. The standardized mortality ratios when the husbands were nonsmokers, exsmokers or smokers of 19 or more cigarettes per day, and smokers of 20 or more cigarettes per day were 1.0, 1.10, and 1.30, respectively (one-sided p for trend, 0.019).

In a Scottish study by Gillis et al. (1984), nonsmokers not exposed to tobacco smoke were compared with nonsmokers exposed to tobacco smoke, with respect to the prevalence of cardiovascular symptoms at entry and mortality due to coronary heart disease. There was no consistent pattern of differences in coronary heart disease or symptoms between nonsmoking men exposed to tobacco smoke and their nonexposed counterparts. Nonsmoking women exposed to tobacco smoke exhibited a higher prevalence of angina and major electrocardiogram (ECG) abnormality at entry, and also a higher mortality rate for all coronary diseases. However, rates of myocardial infarction mortality were higher for exposed nonsmoking men and women compared with the nonexposed nonsmokers. The rates were 31 and 4 per 10,000, respectively, for the nonexposed nonsmoking men and women, and 45 and 12 per 10,000, respectively, for the exposed nonsmoking men and women. None of the differences were tested for statistical significance.

In the Japanese and the Scottish studies, other known risk factors for cardiovascular diseases (i.e., systolic blood pressure, plasma cholesterol) were not taken into account in the analysis.

In a study of heart disease, Garland et al. (1985) enrolled 82 percent of adults aged 50 to 79 between 1972 and 1974 in a predominantly white, upper-middle-class community in San Diego, California. Blood pressure and plasma cholesterol were measured at entry, and all participants responded to a standard interview which asked about smoking habits, history of heart disease, and other health-related variables. Excluding women who had a previous history of heart disease or stroke or who had ever smoked, 695 currently married nonsmoking women were classified by their husbands' self-reported smoking status at enrollment. After 10 years of follow-up, there were 19 deaths due to ischemic heart disease; the age-standardized mortality rates for nonsmoking wives whose husbands were nonsmokers, exsmokers, and current smokers were 1.2, 3.6, and 2.7, respectively (one-sided p for trend, ≤ 0.10). After adjustment for age, systolic blood pressure, total plasma cholesterol, obesity index, and years of marriage, the relative risk for death due to ischemic heart disease for women married to current or former smokers at entry compared with women married to never smokers was 2.7 (one-sided p ≤ 0.10).

The study's findings are not convincing from the point of view of sample stability. The total number of deaths due to ischemic heart disease was small, and the denominator in the relative risk calculation is unstable, based on the deaths of two women whose husbands had never smoked. Moreover, it is well established that the risk of coronary heart disease is substantially lower among those who have stopped smoking (U.S. Department of Health and Human Services, 1984), although the amount of time required for this change after cessation of smoking is not clear (Kannel, 1981). In this study, 15 of 19 deaths occurred in nonsmoking women married to husbands who had stopped smoking at entry, and the age-standardized rate for ischemic heart disease was highest in this group. The high proportion of deaths in nonsmoking women married to men who stopped smoking implies that the excess resulted from sustained effects of involuntary smoking. More detailed characterizations of exposure to ETS and specific types of cardiovascular disease associated with this exposure are needed before an effect of involuntary smoking on the etiology of cardiovascular disease can be established.

One study (Aronow, 1978) suggested that involuntary smoking aggravates angina pectoris. This study was criticized because the end point, angina, was based on subjective evaluation, and because other factors such as stress were not taken into account (Coodley, 1978; Robinson, 1978; Waite, 1978; Wakehan, 1978). More importantly, the validity of Aronow's work has been questioned (Budiansky, 1983).

Both the National Research Council (1986a) and the Surgeon General of the U.S. (1986) concluded that although several studies show excessive risk of cardiovascular disease in ETS-exposed nonsmokers, methodological problems in study design and analysis preclude any firm conclusions on the association between ETS exposure and cardiovascular disease.

Other health effects of ETS which have been examined are primarily related to studies of growth and health of children exposed to ETS. National Research Council (1986a) reviewed these studies, particularly those associated with the influence of ETS exposure on the birthweight of newborn babies born to nonsmoking women exposed to ETS while pregnant, and potential ETS effects on childhood growth, and its role in the excess development of ear infections. All of these studies were unable to differentiate effects of *in utero* exposure, from childhood exposures to ETS.

2.4.5 IAQ Control Options

The obvious methods for the control of exposure of nonsmokers to the sidestream and exhaled mainstream smoke of smokers are:

1. The prohibition of smoking in public places.
2. The elimination of smoking in the workplace where nonsmokers are present or the complete segregation of smokers.
3. The educating of the nonsmoking public (particularly the nonsmoking spouses of smokers) to the hazards of passive smoke.

A public awareness campaign on the part of federal, state, and city governments has had a significant effect in reducing exposure of nonsmokers to SS of smokers, but complete elimination has not been achieved.

Fortunately, other control options are also available:

1. Properly designed air ventilation systems can be effective in reducing the concentration of ETS in the indoor environment, especially if air from smoking areas is exahusted from the building. This however, carries an energy penalty. This may be partly offset through the use of air-to-air heat exchangers.
2. The use of high-efficiency filters on electrostatic devices, either installed in the ductwork of central air handling systems or as portable units, strategically placed, can be effective in removing the PM of ETS. Little is known about the effect of such devices on the gas phase of smoke. Questions have also been raised concerning ozone production by these electrostatic devices.
3. Negative ion generators, when properly built and installed, are said to provide an effective means of removal of smoke constituents. More research is needed to determine removal effectiveness and design parameters.

2.4.6 Conclusions

2.4.6.1 What is Known. Evidence has accumulated indicating that nonsmoking pregnant women exposed to ETS on a daily basis for several hours are at increased risk for producing babies of low birthweight. The mechanisms that reduce birthweight are, as yet, unknown. Recent studies show a dose-response relationship between the number of cigarettes smoked by the father and birthweight of the children of nonsmoking pregnant women.

A few studies have reported that children of smokers have reduced growth and development. These require further corroboration to differentiate *in utero* exposure from subsequent childhood exposures.

Household exposure to ETS is linked with increased rates of chronic ear infections and middle-ear effusions in young children. For children with nasal allergies and recurrent otitis media, ETS exposures may synergistically increase their risk for persistent middle-ear effusions.

2.4.6.2 What Scientific Information is Missing. Experimental studies should be developed to articulate possible mechanisms through which paternal smoking adversely effects fetal growth in nonsmoking pregnant women. Special emphasis should be placed on identifying relevant effects of pregnancy on excretion and absorption of ETS, including transplacental metabolism.

Additional study is needed to corroborate one finding of a dose-response relationship between reduced height of children and increasing numbers of cigarettes smoked in the home, regardless of whether the mother smoked during pregnancy and regardless of which parent smoked.

Research should be conducted to explore the mechanisms by which exposure to ETS might adversely affect the functioning of the ear and to study possible long-term consequences of ETS exposure for the auditory apparatus.

2.4.6.3 Research Needs. The National Research Council report (1986a) on ETS also provides an extensive review of the data available and research needed on the assessment of human exposure and dosimetry of ETS.

The National Research Council and the Surgeon General concluded that laboratory studies can contribute to a better understanding of the factors and mechanisms involved in the induction of cancer and the cancer potency of ETS. There have been numerous bioassays conducted on MS. In examining the effects of MS, many research workers used condensates of the smoke painted on the shaved skin of mice. This contrasts with human exposure, which is mainly to the respiratory tract. Nonetheless, these skin-painting studies have been useful in examining the carcinogenicity of different tobacco constituents and have advanced knowledge of the actions of MS on a gross exposure level. Except for one study which provides suggestive evidence that SS is more tumorigenic than MS, similar work with skin painting has not been done with ETS and would be of value for assessing the differential toxicity and carcinogenicity of ETS and MS.

In contrast to MS exposure, ETS exposure involves proportionately more exposure to gas phase than to particulate phase constituents. There have been no studies of the effects of exposure to aged ETS. The relative *in vivo* toxicities of MS, SS, and ETS need to be assessed.

Some studies have attempted to evaluate the gas phase of MS, SS, and ETS in short-term, *in vitro* assays. A solution of the gas phase of MS has been shown to induce dose-dependent increases in sister-chromatid exchanges in cultured human lymphocytes. Mutagenic activity has been found in the PM of SS and in condensates of ETS. However, the work done to date is too sparse to permit any estimates of the mutagenicity of ETS per se, even though most of ETS consists of SS. Further *in vitro* assays of ETS are needed.

There have been few studies of risk for cancers other than lung in nonsmokers exposed to ETS. Some of the sites considered have been the brain, the hematopoietic system, and all sites combined. The results of these studies have been inconsistent. Whether there is an association between ETS exposure and cancers of any site other than the lung is an important topic for future epidemiologic inquiries.

2.5 NONCOMBUSTION PARTICLES

2.5.1 Asbestos

Asbestos is a generic term that applies to a group of impure hydrated silicate minerals which occur in various fibrous forms (Hawley, 1981). They are incombustible and separable into filaments. Types of asbestos include amphiboles, such as amosite, crocidolite, tremolite, anthophyllite, and actinolite, and chrysotile. Chrysotile, a fibrous form of serpentine, is a magnesium silicate whose fibers are strong and flexible, and its longer fibers can be spun into thread for weaving (Hawley, 1981; Sittig, 1985). It is the most widely used form of asbestos in the United States. Amphibole asbestos includes various silicates of magnesium, iron, calcium, and sodium. Its fibers are generally brittle and cannot be spun, but it is more resistant to heat than chrysotile asbestos (Hawley, 1981).

Asbestos has been used in fireproof fabrics, brake linings, gaskets, roofing compositions, electrical and heat insulations, paint fillers, and chemical filters; as a reinforcing agent in rubber and plastics; and as a component of paper dryer felts and diaphragm cells (Hawley, 1981). Most asbestos has been used in the construction industry. Most (92 percent) of the one-half million tons used in the U.S. is firmly bonded in such products as floor tiles, asbestos cements, roofing felts, and shingles. The rest is friable or in powder form, present in insulation materials, asbestos cement powders, and acoustical products (Sittig, 1985). Inhalation of free fibers can result in a fibrosis of the lung known as asbestosis, and in the development of mesotheliomas, and cancers of the lung and gastrointestinal tract.

2.5.1.1 Sources. While no longer installed in homes or commercial buildings, asbestos was widely used as a constituent of ceiling tiles and for pipe, duct, and attic insulation. The typical size of asbestos fibers is 0.1 to 10 μm in length, and when disturbed, asbestos fibers may become suspended in the air for many hours, thus increasing the extent of asbestos exposure for individuals within the area (U.S. Environmental Protection Agency, 1985c). Fallout from asbestos installations may occur without overt physical disruption of the fiber-bearing material and may simply be a function of adhesive degradation, vibration, humidity variations, air movement from heating and ventilating equipment, and air turbulence and vibration caused by human activity (U.S. Environmental Protection Agency, 1978). Usually, asbestos fibers released in buildings have been associated with visible damage or erosion of asbestos materials such as insulation, asbestos cement piping and insulation, floor tiling, shingles, or other asbestos-containing material. Many buildings with such materials intact have no increased concentrations of asbestos in air.

During 1974, 116 samples of indoor and outdoor air were collected in 19 buildings (usually 4 to 6 indoor samples and 1 ambient air control sample per building) in 5 U.S. cities to assess whether contamination of the building air resulted from the presence of asbestos-containing surfacing materials in rooms or return air plenums (Nicholson et al., 1975). The asbestos materials in the buildings were of two main types: 1) a cementitious or plaster-like material that had been sprayed as a slurry onto steelwork or building surfaces, and 2) a loosely bonded fibrous mat that had been applied by blowing a dry mixture of fibers and binders through a water spray onto a target surface. The friability of the two types of materials differed considerably; the cementitious spray surfaces were relatively impervious to damage while the fibrous sprays were highly friable. The results of air sampling in these buildings provide evidence that the air of buildings with fibrous asbestos-containing materials may often be contaminated.

Weathering of asbestos cement wall and roofing materials was shown to be a source of asbestos air pollution by analyzing air samples taken in buildings constructed of such material (Nicholson, 1978). Seven samples taken in a school after a heavy rainfall showed asbestos concentrations from 20 to 4500 ng/m^3 (arithmetic mean = 780 ng/m^3); all but two samples exceeded 100 ng/m^3. The source was attributed to asbestos washed from asbestos cement walkways and asbestos cement roof panels. No significantly elevated concentrations were observed in a concurrent study of houses constructed of asbestos cement materials. Roof water runoff from the homes landed on the ground and was not reentrained, while that of the schools fell to a smooth walkway, which allowed easy reentrainment when dry. Contamination from asbestos cement siding has also been documented by Spurny and co-workers (1980).

Asbestos may enter buildings from outside sources. One of the most significant remaining contributions to environmental asbestos concentrations may be emissions from braking of automobiles and other vehicles. Measurements of brake and clutch emissions reveal that, annually, 2.5 tons of unaltered asbestos are released to the atmosphere, and an additional 68 tons fall to roadways where some of the asbestos is dispersed by passing traffic (Jacko et al., 1973). Infiltration of outside air, or entry through building air intakes may disperse asbestos fibers from these sources indoors.

Asbestos flooring is used in a large number of buildings and is the third largest use of asbestos fibers. Sebastien et al. (1982) measured concentrations of indoor airborne asbestos up to 170 ng/m^3 in a building with weathered asbestos floor tiles.

2.5.1.2 Monitoring. The analysis of ambient air samples for asbestos has utilized techniques different from those used in occupational circumstances. This situation occurred because typical urban air may contain up to 100 μg/m^3 of particulate matter in which the researcher is attempting to quantify asbestos concentrations from about 0.1 ng/m^3 to perhaps 1000 ng/m^3. Thus, asbestos may constitute only 0.0001 to 1 percent of the particulate matter in a given air sample. Asbestos found in ambient air has a size distribution such that the vast majority of fibers are too short or too thin to be seen with an optical microscope. In many cases, these fibers and fibrils will be agglomerated with a variety of other materials present in the air samples.

The only effective method of analysis uses electron microscopy to enumerate and size all asbestos fibers (Nicholson and Pundsack, 1973; Samudra et al., 1978). Samples for such analysis are usually collected either on a Nuclepore® (polycarbonate filter with a pore size of 0.4 μm or on a Millipore® (cellulose ester) filter with a pore size of 0.8 μm. In some cases the Millipore® is backed by nylon mesh. Samples collected on Nuclepore® filters are prepared for direct analysis by carbon coating the filter to entrap the collected particles. A segment of the coated filter is then mounted on an electron microscope grid, which is placed on a filter paper saturated with chloroform so that the chloroform vapors dissolve the filter material. (Earlier electron microscopic analysis utilized a rub-out technique in which the ash residue was dispersed in a nitrocellulose film on a microscope slide and a portion of the film was then mounted on an electron microscope grid for scanning.)

Samples collected on Millipore® filters are prepared for indirect analysis by ashing a portion of the filter in a low-temperature oxygen furnace. This removes the membrane filter material and all organic material collected in the sample. The residue is recovered in a liquid phase, dispersed by ultrasonification, and filtered on a Nuclepore® filter. The refiltered material is coated with carbon and mounted on a grid as described above. The samples are then subjected to analysis. Chrysotile asbestos is identified on the basis of its morphology as seen with the electron microscope, and amphiboles are identified on the basis of morphology under electron microscopy and by their selected area electron diffraction patterns, supplemented by energy-dispersive X-ray analysis. Fiber concentrations in fibers per unit of volume (such as fibers/cm^3, fibers/m^3, etc.) are calculated based on sample volume and filter area counted. In some cases, mass concentrations are reported using fiber volume and density relationships. However, mass concentrations may not be reliable if the sample contains fibrous forms, such as clusters, bundles, and matrices, in which fiber volume is difficult to determine. These materials may constitute most of the asbestos mass in some samples, particularly those reflecting emission sources. Current fiber counting methods do not include those clumps. However, many of them are respirable and to the extent that they are broken apart in the lungs into individual fibers, they may add to the carcinogenic risk. On the other hand, methods that break up fibers generally disperse the clumps as well. In such analyses, the clumps would contribute to the mass.

In much of the earlier analyses of chrysotile concentrations in the United States, the ashed material was either physically dispersed or disrupted by ultrasonification. Thus, no information was obtained on the size distribution of the fibers in the original aerosol. Air concentrations were given only in terms of total mass of asbestos present in a given air volume, usually nanograms per cubic meter (ng/m^3). With the use of Nuclepore® filters and appropriate care in the collection of samples and their processing, information on the fiber size distribution can be obtained, and concentrations of fibers of selected dimensions can be calculated. Samples collected on Millipore® filters can be ashed and the residue resuspended and filtered through Nuclepore® filters. However, some breakage of fibers for electron microscopic analysis has been reported by Burdett and Rood (1983) and is being tested by several laboratories. However, the utility and reliability of this technique is unknown at present.

Current measurements of low-level contamination with asbestos use electron microscope techniques, which determine the total mass of asbestos present in a given volume of air. Previous measurements of concentrations of fibers longer than 5 µm were made using optical microscopy, or from optical microscopy of total particulate samples. Occupational studies used the latter techniques. If information regarding health effects from these studies is to be extrapolated to measurements made in nonindustrial indoor spaces, a relationship between optical fiber counts and mass of asbestos determined by electron microscopy must be established. Crude estimates of a conversion factor relating fiber concentration in fibers per milliliter (f/ml) to airborne asbestos in micrograms per cubic meter ($\mu g/m^3$) is derived from several studies, and is detailed in the Airborne Asbestos Health Update (Nicholson, 1986). These crude conversion factors relating mass concentrations to optical fiber concentrations range from 5 to 150, and by necessity introduces large uncertainties. For extrapolation of low-mass concentrations the geometric mean to fiber count of the above range of conversion factors, which is 30 $\mu g/m^3/f/ml$ is used in the EPA asbestos health assessment (U.S. Environmental Protection Agency, 1986). The geometric standard deviation of this value is 4, and this uncertainty severely limits any extrapolation in which it is used. In the case of amosite, the data of Davis et al. (1978) suggest that a conversion factor of 18 is appropriate. However these data yield lower chrysotile values than all other chrysotile estimates; therefore, they may also be low for amosite (Nicholson, 1986).

2.5.1.3 Exposure. Measurements using electron microscopy techniques established the presence of asbestos in the urban ambient air, usually at concentrations less than 10 ng/m^3. Concentrations of 100 ng/m^3 to 1000 ng/m^3 were measured near specific asbestos emission sources, in schools where asbestos-containing materials are used for sound control, and in office buildings where similar materials are used for fire control. The value of 300 ng/m^3 corresponds to about 10,000 fibers/m^3, but this is a crude estimate. Excess concentrations in buildings have usually been associated with visible damage or erosion of the asbestos materials. Many buildings with intact material have no increased concentrations of asbestos. Most ambient measurements were taken over ten years ago, therefore it is very important to obtain more current data. Asbestos exposure data pertain essentially to conspicuous episodes; the studies were not designed to provide representative measures of ambient concentrations throughout the United States.

Table 2-29 summarizes those studies of the general ambient air or of specific pollution circumstances that have a sufficient number of samples for comparative analysis. The data are remarkably consistent. Average 24-hour samples of general ambient air indicate asbestos concentrations of 1 to 2 ng/m^3 (two U.S. samples that may have been affected by specific sources were not included). Short-term daytime samples are generally higher, reflecting the possible contributions of traffic, construction, and other human activities. In buildings having asbestos surfacing materials, average concentrations 100 times greater than ambient air are seen in some schools, and concentrations 5 to 30 times greater than that in ambient air are seen in some other buildings.

Of concern was the discovery of extensive asbestos use in public school buildings (Nicholson et al., 1978). Asbestos surfaces were found in more than 10 percent of pupil-use areas in New Jersey schools, with two-thirds of the surfaces showing some evidence of damage. Because these values appear to be typical of conditions in many other states, it was estimated that 2 to 6 million pupils and 100,000 to 300,000 teachers may be exposed to released asbestos fibers in schools across the nation. To obtain a measure of contamination for this use of asbestos, 10 schools were sampled in the urban

TABLE 2-29. SUMMARY OF ENVIRONMENTAL ASBESTOS SAMPLING STUDIES

Sample Set	Collection Period	Number of Samples	Mean Concentration ng/m^3
Quarterly composites of 5 to 7 24-hr U.S. samples (Nicholson, 1971; Nicholson and Pundsack, 1973)	1969-70	187	3.3 C[a]
Quarterly composite of 5 to 7 24-hr U.S. samples (U.S. Environmental Protection Agency, 1974)	1969-70	127	3.4 C
5-day samples of Paris, France (Sebastien et al., 1980)	1974-75	161	0.96 C
6- to 8-hour samples of New York City (Nicholson et al., 1971)	1969	22	16 C
5-day, 7-hour control samples for U.S. school study (Constant et al., 1983)	1980-81	31	6.5 (6C, 0.5A[b])
16-hour samples of 5 U.S. cities (U.S. Environmental Protection Agency, 1974)	1974	34	13 C
New Jersey schools with damaged asbestos surfacing materials in pupil use areas (Nicholson et al., 1978)	1977	27	217 C
U.S. school rooms/areas with asbestos surfacing material (Constant, 1983)	1980-81	54	183 (179C, 4A)
U.S. school rooms/areas in building with asbestos surfacing material (Constant, 1983)	1980-81	31	61 (53C, 8A)
Buildings with asbestos materials in Paris, France (Sebastien et al., 1980)	1976-77	135	35 (25C, 10A)
U.S. buildings with friable asbestos in plenums or as surfacing materials (Nicholson et al., 1975; Nicholson et al., 1976)	1974	54	48 C
U.S. buildings with cementitious asbestos material in plenums or as surfacing materials (Nicholson et al., 1975, 1976)	1974	28	15 C
Ontario buildings with asbestos insulation (Ontario Royal Commission, 1984)	1982	63	2.1

[a] C = Chrysotile

[b] A = Amphibole

centers of New York and New Jersey and in suburban areas of Massachusetts and New Jersey. Schools were selected for sampling because of visible damage, in some cases extensive.

Samples were taken over 4 to 8 hours in 10 schools (1 to 5 samples per school). Chrysotile asbestos concentrations ranged from 9 ng/m^3 to 1950 ng/m^3, with an average of 217 ng/m^3. Outside air samples at 3 of the schools varied from 3 ng/m^3 to 30 ng/m^3, with an average of 14 ng/m^3. In all samples but two (which measured 320 ng/m^3) no asbestos was visible on the floor of the sampled area, although surface damage was generally present near the area. The highest value (1950 ng/m^3) was in a sample that followed routine sweeping of a hallway in a school with water damage to the asbestos surface, although no visible asbestos was seen on the hallway floor. It is emphasized that the schools were selected in testing on the basis of the presence of visible damage. Although the results cannot be considered typical of all schools having asbestos surfaces, the results do illustrate the extent to which contamination can exist.

A recent study suggests that the above school samples may not be atypical (Constant et al., 1983). Concentrations similar to those indicated above were found in the analysis of samples collected during a 5-day period in 25 schools that had asbestos surfacing materials. The schools were in a single district and were selected by a random procedure, not because of the presence or absence of damaged material. A population-weighted arithmetic mean concentration of 179 ng/m^3 was measured in 54 samples collected in rooms or areas that had asbestos surfacing material. In contrast, a concentration of 6 ng/m^3 was measured in 31 samples of outdoor air taken at the same time. Of special concern are 31 samples collected in the schools that used asbestos, but taken in areas where asbestos was not used. These data showed an average concentration of 53 ng/m^3, indicating dispersal of asbestos from the source. As published fiber counts were fibers of all sizes, only the fiber mass data are listed in the table. Additionally, fiber clumps were noted in many samples, but were not included in the tabulated masses.

2.5.1.4 Health Effects. The most serious health effects from asbestos exposure are lung cancer and mesothelioma (cancer of tissue of mesothelial origin), and these have been established as the most important causes of death from asbestos exposure. While the relationship of occupational exposure to asbestos with these cancers has been established from occupational/epidemiologic studies, the occurrence of asbestosis has been documented more extensively than the risk of the asbestos-related malignancies. In part, this documentation resulted from knowledge of this disease extending back to the turn of the century, whereas the malignant potential of asbestos was not suggested until 1935 (Lynch and Smith, 1935; Gloyne, 1936) and not widely appreciated until the 1940s (Merewether, 1949). Asbestosis had been documented in a wide variety of work circumstances and associated with all commercial types of asbestos fibers. Among some heavily exposed groups, 50 to 80 percent of individuals employed for 20 or more years in asbestos-associated industries were found to have abnormal X-ray films characteristic of asbestos exposure (Selikoff et al., 1965; Lewinsohn, 1972). A lower percentage of abnormal X-ray films was present in lesser exposed groups. Company data supplied to the British Occupational Hygiene Society (BOHS) (British Occupational Hygiene Society, 1968) on X-ray and clinical abnormalities among 209 employees of a large textile production facility in Great Britain were analyzed by Berry (1973) in terms of a fiber exposure-response relationship. The results were utilized in establishing the 1969 British regulation on asbestos. These data suggested that the risk of developing the earliest signs of asbestosis (rales) was less than 1 percent for accumulated fiber exposure of 100 fiber-years/ml (f-y/ml), (e.g., 2 fibers/milliliter (f/ml) for 50 years). However, shortly after the establishment of the British standard, additional data from the same factory population suggested a much greater prevalence of X-ray abnormalities than was believed to exist at the time the British standard was set (Lewinsohn, 1972). These data resulted from use of the new International Labour Office (ILO) U/C standard classification of X-rays (International Labour Office, 1971) and the longer time from onset of employment. Of the 290 employees whose clinical data were reviewed by the BOHS, only 13 had been employed for 30 or more years and 172 had less than 20 years of employment. The progression of asbestosis depends on both cumulative exposure and time from exposure; therefore, analysis in terms of only one variable can be misleading.

Extrapolations of risks of asbestos cancers from occupational circumstances have been made, although numerical estimates in a specific exposure circumstance have a large (approximately tenfold) uncertainty. Because of this uncertainty, calculations of unit risk values for asbestos at the low concentrations measured in the environment must be viewed with caution. The best estimate of risk to the United States general population for a lifetime continuous exposure to 100 fibers/m^3 is 28 mesothelioma deaths and 5 excess lung cancer deaths per million females. Corresponding numbers for males are 19 mesothelioma deaths and 17 excess lung cancer deaths per million individuals. Excess gastrointestinal (GI) cancer mortality is approximately 10 to 30 percent that of excess lung cancer mortality. These risks are subjective, to some extent, and are also subject to the following limitations in data: (1) variability in the exposure-response relationship at high exposures; (2) uncertainty in extrapolating to exposures 1/100 as much; and (3) uncertainties in conversion of optical fiber counts to electron microscopic fiber counts or mass determinations (Nicholson, 1986).

2.5.2 Dusts, Sprays, and Cooking Aerosols

2.5.2.1 Introduction. House dust potentially can be a significant source for the intake of toxic materials through both inhalation and ingestion, especially for young children who spend much of their time on the floor inside homes (Roberts et al., 1987). Dust can be a medium for the transfer of toxics from sources in and outside the house to people and a medium for the reservoir of toxics, especially pesticides. An estimate of average daily soil dust ingestion by small children ranges from 0.12 to 1.8 grams (Binder et al., 1986). The composition of house dust is highly variable, and includes residues from food and food preparation; hair and skin scale from humans and animals; fibers from clothing, household furnishings, and building materials; aerosols from cleaning compounds, waxes, and other consumer products; fragments of vegetation and humus; and mineral particles. Van Houdt and Boleij (1984) reported that the indoor airborne particulate matter collected in homes (living rooms and kitchens) was more mutagenic than the samples collected outdoors. Studies by Roberts et al. (1987) showed that the dust samples (50 mesh fractions) collected from the rugs in homes by vaccum cleaners were positive in Ames *Salmonella* mutation assay and *E. coli* DNA repair assay.

During the process of cooking, carcinogenic compounds such as PAHs, (parent compounds), nitro-PAHs, and heterocyclic amines have been found. These compounds can be emitted in the air, contained or adsorbed onto the food surfaces. Lijinski and Shubik (1964) first reported the appearance on the benzo(a)pyrene and other PAH compounds on the surface of charcoal-broiled beef steaks. Highly mutagenic and carcinogenic heterocyclic amines have been isolated from cooked food, pyrolysates of amino acids and proteins, and the heated mixtures of creatinine, sugars, and amino acids (Sugimura, 1985). Formation of these compounds is highly temperature-dependent. Ohnishi et al. (1984) reported the mutagenic activity of chicken and the detection of 1-nitropyrene in the meat. Relatively few studies have assessed the health effects of air emissions during cooking. Lewtas et al. (1987) reported mutagenic activity in kitchens in a pilot home study. As to the health effects of sprays used in homes, little information is available.

2.5.2.2 Monitoring. The need for a sampling device that would collect and maintain the integrity of organic chemicals on house dust was recognized when the U.S. EPA's Total Exposure Assessment Methodology (TEAM) studies were planned in 1978. The absence of such a sampler, however, resulted in the elimination of such monitoring from the TEAM program. The need still exists and is important to the THE effort. Consequently, initial efforts were begun in early 1987 to develop a sampler that will combine the features of a home vacuum cleaner and air sampler, such as that used for high-volume sampling of pesticides and related SVOCs (Lewis and Jackson, 1982). Further work needs to be conducted to test the prototype device and refine it as necessary.

2.5.3 IAQ Control Options

The resuspension of particulate matter using common vacuum machines has been studied and tests have indicated that a sizeable portion of fine particulate matter escapes the bags used for collection. The development of a more efficient vacuum cleaning method for household and office use would be helpful.

Periodic cleaning of furnace and air conditioning filters is helpful in removing the larger particles. Negative ion generators have been shown to be helpful in removing particulate matter but their performance is highly variable among different manufacturers.

The U.S. EPA has issued guidelines for the removal and disposal of asbestos (U.S. Environmental Protection Agency, 1978). Asbestos-containing materials that are exposed to indoor air should either be covered to prevent fibers getting into the air, or removed.

Exposure to fine liquid aerosols from various consumer "aerosol" spray products can be controlled best by avoiding use of such products. Use under well ventilated conditions is the minimum prudent option. Face masks designed for dust are ineffective for such aerosols. Masks designed for organic vapor control will be partially effective, but may let a substantial fraction of submicron particles through. Research on such devices might lead to improved, and highly effective, designs.

The use of efficient range hoods is advocated during cooking operations. Currently, however, there is little information on the effectiveness of range hoods in removing vapor phase compounds, aerosols, and odors from cooking. This is an area needing investigation.

2.6 NONCOMBUSTION GAS-PHASE ORGANIC COMPOUNDS

2.6.1 Gas-Phase Organic Compounds (Volatile Organic Compounds)

2.6.1.1 Introduction. As many as 300 organic compounds were identified in homes during the TEAM study conducted by the U.S. Environmental Protection Agency (Wallace et al., 1986). These compounds can originate as combustion products or can be emitted from building materials and household chemicals. It is conceivable that several to many may be present in concentrations that could be of concern. Each individual compound has physical and chemical properties that in themselves may cause it to have interactions with biological systems as well as effects on materials. Some of the compounds have been identified as irritants and/or neurotoxicants, and some can act as carcinogens, cocarcinogens, or promoters of cancer in animals and/or humans (Ammann et al., 1986).

2.6.1.2 Occurrence and Sources of Gas-Phase Organic Compounds. Various studies of indoor air quality have identified more than 250 different organics at levels greater than 1 ppb (Sterling, 1985). Many hundreds of additional compounds undoubtedly exist at lower levels. Individual researchers commonly measure 30 to 50 separate compounds (De Bortoli et al., 1986; Lebret et al., 1984; Molhave, 1982; Seifert et al., 1986). An evaluation of the data reported in these and other studies indicates the following:

1. an extremely wide variety of organic compounds are found in the indoor environment
2. the range of measured concentrations between different organic compounds is extremely wide, often two or more orders of magnitude; also, the range of concentrations for a specific compound can vary widely between measurements

3. in most cases, the concentrations of specific organic compounds exceeds the outdoor concentration, thus indicating that the source of the compound is indoors

4. the sources of the organic compounds are quite numerous within any indoor environment; the sources vary depending on the type of building studied

While the available IAQ data present a complex picture regarding organic vapors, there is substantial evidence relating indoor occurrence to emission sources.

2.6.1.2.1 Outdoor sources of volatile organic compounds. Dozens of organic compounds have been found in outdoor air and in the exhaled breath of individuals exposed to these compounds (Wallace, 1986b). Many organic compounds are also contained in water supplies in sufficient quantity to provide most of the observed exposure to such compounds. Water has been shown to provide nearly all of the exposure to three brominated trihalomethanes and to chloroform (Wallace, 1986b).

2.6.1.2.2 Sources of indoor organic vapors. Many indoor materials, including paints, stains, adhesives, and caulks, contain petroleum-based solvents. Such solvents are comprised of a variety of organic compounds often found in indoor environments. All general surveys of indoor air quality include the detection of solvent-based compounds, and there is consistency between the surveys in the detection of specific organics. For example, Molhave (1982) identified 22 compounds of the same organic species, among the 52 compounds emitted from 42 common materials used in Denmark, that were also identified by Seifert et al. (1986) from among 43 compounds detected in 159 samples from public buildings in West Germany. Chlorinated solvents are also used in a wide variety of consumer products and are commonlyfound in indoor air. For example, an EPA study by Steele (1985) determined the chlorocarbon content of more than 1100 household products, including shoe polishes, water repellents, cleaning fluids, epoxy paint sprays, brush cleaners, primers, stains, and varnishes. Table 2-30 indicates the variety of compounds which are emitted from widely used solvents. This table is meant to illustrate the complexity of the organic emissions from solvents in indoor materials; it is not an exhaustive list of all solvent-based organics. It should be noted that many of the compounds in Table 2-30 are not emitted exclusively from solvents. For example, gasoline vapors from attached garages and stored fuel may contribute significant quantities of benzene,

TABLE 2-30. SOLVENT BASED ORGANIC COMPOUNDS

Compound Class	Most Commonly Found Compounds
n-Alkanes	C-9 (Nonane), C-10 (Decane), C-11 (Undecane)
Isoalkanes	C-9 (Isooctane), C-10, C-11
Cycloalkanes	C-6 (Cyclohexane), C-7, C-8, C-9
Aromatics	C-6 (Benzene), C-7 (Toluene), C-8 (Xylene), C-9 (Trimethylbenzene)
Ketones	C-3 (Acetone), C-4 (MEK)
Alcohols	C-3 (n-Propanol, iso-Propanol), C-4 (n-Butanol)
Esters	C-4 (Ethylacetate), C-6 (Butylacetate)
Aldehydes	C-5 (n-Pentanal), C-6 (n-Hexanal)
Terpenes	C-10 (Limonene)
Chlorinated Hydrocarbons	C-1 (Carbon Tetrachloride), C-2 (Dichloroethane, Trichloroethane, Dichloroethylene, Trichloroethylene, Perchloroethylene)

xylene, and other hydrocarbons; outgassing from chlorinated water is a source of trichloroethylene and other halogenated organics (Andelman, 1985); and perchloroethylene is emitted from dry-cleaned clothes (Wallace et al., 1984a,b,c).

While solvent-based indoor emissions come from an extremely wide variety of materials and products, other organic vapors can be associated more closely with specific sources. Table 2-31 lists specific compounds and their associated sources. As with Table 2-30, this table is not all inclusive, but illustrates a variety of organic vapor sources.

2.6.1.2.3 Emission rates. To determine the impact of indoor material/product sources on indoor concentrations of organic vapors, the emission rates for the various compound/source combinations are required. To date, such emission rate data have been developed only for formaldehyde from pressed wood products. The

TABLE 2-31. SPECIFIC INDOOR SOURCES OF ORGANIC VAPORS

Compound	Material Source(s)	Reference
Paradichlorobenzene	Moth crystals, Room deodorants	Nelms et al. (1987)
Methylene Chloride	Paint removers	Girman and Hodgson (1986)
Chloroform	Chlorinated water	Wallace (1986b)
Formaldehyde	Pressed wood products, Foam insulation (UFFI), Textiles, Disinfectants	National Research Council (1981)
Styrene	Plastics, Paints	Wallace (1986b)
Toluene Diisocyanate	Polyurethane foam aerosols	Carroll et al. (1976)
Phthalic Acid Anhydride, Trimellitic Acid, Triethylene Tetraamine	Epoxy resins	Fawcett et al. (1977)
Sodium Dodecyl Sulfate	Carpet shampoo	Kreiss et al. (1982)
Benzyl Chloride, Benzal Chloride	Vinyl tiles plasticized with butyl benzyl phthlate	Rittfeldt et al. (1984)
Ethylene Oxide	Sterilizers (Hospitals)	

U.S. Department of Housing and Urban Development (HUD) has developed standards for formaldehyde emissions from pressed wood products used in mobile homes (Federal Register, 1984). These standards involve chamber testing of materials to determine compliance with emission restrictions. As part of the HUD regulation, the Berge equation is used to evaluate the chamber test results. The Berge equation relates formaldehyde concentration at standard conditions (e.g., 25°C, 50 percent relative humidity) to the test chamber concentration at different values of temperature and relative humidity (Godish and Rouch, 1985). The Berge equation predicts increasing emissions with increasing temperatures and decreasing relative humidity. Matthews (1986) and his co-workers at Oak Ridge National Laboratory have developed other models for determining formaldehyde emission rates which incorporate the following variables: temperature, relative humidity, air exchange rate, product loading, and formaldehyde concentration in the chamber. Matthews' models show that emission rates increase with increases in air exchange rate, decreases in product loading, and decreases in chamber concentration. EPA's Air and Energy Engineering Research Laboratory conducted a cooperative project with Matthews to compare small chamber testing procedures for determining emissions from particleboard. Table 2-32 provides emission rates for formaldehyde and other organics for various test conditions (Nelms et al., 1986). All tests were conducted at 23°C and 50 percent relative humidity on particleboard aged approximately eight months. The emission rates in the bottom row of Table 2-32 are representative of air exchange rates and loadings found in residential environments. Material newer than eight months could be expected to have higher rates. The material tested was low-density particleboard normally used in home construction (e.g., flooring material). Medium-density particleboard, such as is used in furniture, has formaldehyde emission rates two to four times higher (Matthews, 1986). Plywood products generally have lower emission rates than particleboard. Note that the formaldehyde emission rates are related to the air exchange and loading relationships discussed above. The emission rates for the other organic compounds do not vary substantially with these two parameters and appear to be limited by the rate of diffusion to the surface of the particleboard.

TABLE 2-32. EMISSION RATES FROM PARTICLEBOARD (μg/m^2 hr)

Air Exchange (hr^{-1})	Loading (m^2/m^3)	Formaldehyde	Acetone	Hexanal	Others*
2.71	1.96	154	37	15	27
0.54	1.17	95	41	26	26
3.61	0.78	230	38	20	31
0.54	0.39	140	37	24	27

*The sum of emission rates for propanol, butanone, benzaldehyde, and benzene.

Limited research has been conducted on the emission rates of organics from materials other than pressed wood products. Molhave (1982) conducted emission rate tests on 42 materials using a one cubic meter test chamber with a ventilation rate of one air change per day. Girman et al. (1984) evaluated emissions from 15 adhesives using 3.8-liter cans with an air exchange rate of 14 per hour. EPA has conducted emission tests on caulk, adhesive, floor wax, and wood stain using a 166 liter chamber at various air exchange rates (Tichenor et al., 1986). The Saskatchewan Research Council is conducting emissions testing of caulking compounds. Georgia Tech Research Institute conducts chamber testing of building materials.

It is difficult to evaluate the limited data on emission rates of organic vapors from indoor materials/products because standard testing protocols have not been developed. A number of test conditions are critical to an effective determination of emission rates, including: temperature, relative humidity, air exchange rate, and product loading (area of sample/volume of test chamber). In addition, the effect of chamber concentration and chamber "wall effects" must be determined. Finally, the age or condition of the sample affects emission rates. Any emission rate data must be coupled with a clear description of all of the variables and phenomena in order to evaluate and compare such data. To show the variability in reported emission rates, Table 2-33 compares emission rate data for two materials based on tests by three investigators. No judgment can be made as to which of the values in the table are the "right answers" without a rigorous evaluation of the variables discussed above.

2.6.1.3 Monitoring of Gas-phase Organics. Previous total human exposure studies (Pellizzari et al., 1982; Wallace, 1986b) have indicated that indoor air concentration of many or most volatile organic chemicals are substantially higher than outdoor levels and constitute a major route of exposure to these chemicals. The principal VOCs of concern are halogenated aliphatic and aromatic hydrocarbons and benzene and its homologs. Sources of these VOCs within residences include building materials and furnishings, cleaning solvents, fuels, and releases from tap water (especially during showering). Many halogenated and benzene family VOCs are known or suspected human carcinogens.

Nearly all monitoring studies for VOCs have depended upon collection on Tenax®-GC sorbent tubes. This sorbent is known to suffer from many problems associated with artifact formation (Walling et al., 1986), very limited capacity for the more volatile compounds (e.g., vinyl chloride and methylene chloride), and for the polar organics (e.g., acrylonitrile and ethylene oxide). Background contamination of the Tenax® is variable from batch to batch. Particular problems occur with toluene, benzene, and to a lesser extent, styrene. Occasional high background contamination and variabilities are encountered with

TABLE 2-33. COMPARISON OF ORGANIC EMISSION RATES: SILICONE CAULK AND FLOOR ADHESIVE

Investigator	Chamber Volume (liter)	Air Exchange Rate (per hr)	Time* (hr)	Emission Material	Factor (mg/m^2 hr)
Girman et al. (1984b)	3.8	14	(1)	Floor Adhesive	140 - 180
Molhave (1982)	1000	0.04	(2)	Floor Adhesive	271
"	"	"	"	Silicone Caulk	26
Tichenor and Mason (1986)	166	1.8	0.5	Floor Adhesive	1700
"	"	"	1	"	700
"	"	"	5	"	100
"	"	"	1	Silicone Caulk	20
"	"	"	5	"	1.6

*The time elapsed since the sample was applied to the substrate.

(1) The sample was dried for 9 - 14 days prior to testing.

(2) The sample was brought to equilibrium prior to testing.

chloroform and 1,1,1-trichloroethane. Careful preparation and cleanup procedures, coupled with extensive precautions during transport and storage, are necessary to insure high quality data.

Despite the limitations of Tenax®, a monitoring system was successfully employed during 1979-85 in the EPA TEAM studies involving 600 residents in four states (Wallace, 1986b). Battery-operated pumps capable of 12-hour continuous operation at 30 cm^3/min flow rate were used to sample air through a cartridge containing 2 g of Tenax-GC. The pump and cartridge were carried in a specially-designed vest worn by the participants. The sampling system was found to be highly acceptable to the participants and proved to be very reliable (less than 1 percent of samples were lost due to pump failure). Approximately twenty VOCs were monitored with sensitivities in the 10 to 100 ng/m^3 range and mean relative standard deviations (RSD) in the 25 to 35 percent range (exception: benzene, percent/RSD = 45 percent).

SVOCs from combustion are usually collected in the particulate phase in which they contribute to the mass loading as RSP. They can be identified and quantified (subject to errors discussed later) using extraction techniques followed by analyses of the extracted material. Some of these materials are volatile and will evaporate from the filter during a 24-hour collection. Unless an adsorbent tube is placed in series behind the filter this material will be lost (Jacob et al., 1986). As described above for RSP, VOCs and SVOCs are heterogeneous and variable mixtures that have component toxicities ranging from inert to toxic or carcinogenic. Indoor VOCs have been measured as concentrations by Lebret et al. (1986), Seifert et al. (1984), and De Bortoli et al. (1984), and as nominal exposures by Wallace et al. (1986). The TEAM study data represent true indoor nominal exposures since the subject carrying the monitor spent 12 hours indoors (Wallace et al., 1986). These data represent 12-hour averages for a sample of subjects in a given city during a specific season. For instance, the arithmetic mean is robust with the averaging time, but both the variance of these data and the higher percentile values will decrease with averaging time, so that the percentiles of indoor exposure that are greater than these values will be much less. For example, Lebret et al. (1986) report data on four homes in a time series of alternating weekly average indoor concentrations, and it can be seen that the variance of these values is considerably less than the TEAM study values. Because these data contain time periods when the home was vacant, they should not be compared directly with the TEAM data, even when allowing for the difference in analytical techniques. Table 2-34 gives the ranges of 12-hour averages, but for interpretation in relation to possible irreversible effects of the carcinogenic materials in the mixture, the annual average exposures need to be predicted.

A passive monitor for VOCs was developed during 1983-85 (Lewis et al., 1985; Coutant et al., 1985). This device is a small, stainless steel cylinder (3.8 cm oc x 1.2 cm), weighs 36 g, and contains a small bed of Tenax®-GC (0.4 g) or other sorbent. The dual-faced device is thermally desorbable and has very high efficiency. Effective sampling rates of 75 to 100 cm^3/min for most VOCs permit detection limits of 1 ppbv or less with exposure times of 1 to 3 hours. Problems due to reversible sorption, which is analogous to break-through in active samplers, arise at loadings greater than 1 μg of VOC (usually after several hours exposure for the more volatile VOCs). Mathematical corrections can be applied if break-through volumes are known, however. The device

TABLE 2-34. WEIGHTED 12-HOUR MEASUREMENTS FOR BREATH (μg/m³) OF RESIDENTS FOR BAYONNE AND ELIZABETH, NJ, COMBINED-TEAM FIRST SEASON

Estimated Population Size:	128,603
Minimum Sample Size:	295
Maximum Sample Size:	339

Compound	Mid Q.L.[a]	Arith. Mean	S.E.[b]	Geo Mean[c]	S.E.[d]	Median	Percentiles 75	90	95	99	Range
Chloroform	2.08	3.12	0.34	1.30	1.13	1.80	3.70	8.20	11.5	26.0	.05-29.0
1,1,1-Trichloroethane	2.30	15.0	2.57	4.79	1.13	6.60	13.0	30.0	42.0	185	.06-520
Benzene	0.44	18.7	1.40	8.19	1.22	12.0	24.0	42.0	56.0	120	.02-200
Carbon Tetrachloride	1.70	1.31	0.26	0.60	1.15	0.69	1.06	2.25	2.7	20.0	.05-250
Trichloroethylene	1.50	1.77	0.21	0.93	1.13	0.88	1.80	3.94	5.9	14.0	.08-30.0
Tetrachloroethylene	4.10	13.3	1.83	7.33	1.08	6.80	12.9	31.0	44.0	190	.12-280
Styrene	0.97	1.15	0.13	0.72	1.11	0.79	1.25	2.40	3.0	7.2	.06-31.0
m,p-Dichlorobenzene	1.32	8.10	1.54	1.72	1.12	1.30	3.50	21.0	44.0	110	.11-158
Ethylbenzene	0.40	4.58	0.55	2.45	1.14	2.90	5.30	8.90	12.0	29.0	.02-290
o-Xylene	1.10	3.35	0.36	1.99	1.12	2.20	3.70	6.30	9.2	17.0	.05-220
m,p-Xylene	0.52	8.95	0.93	5.34	1.12	6.35	11.0	19.0	21.0	53.0	.05-350

[a] Mid Q.L. = Median Quantifiable Limit

[b] S.E. = Standard Error of Arithmetic Mean

[c] Geo. Mean = Geometric Mean

[d] S.F. = Geometric Standard Error = exp(s) where s is the standard error of the weighted mean of log(x).

Source: Wallace et al. (1986).

was modified to reduce the effective sampling rate by a factor of 20 to 25 to permit 24-hr exposures for nearly all VOCs. A high-rate version is commercially available from Scientific Instrument Specialists.

Because of increasing concerns over the reliability of Tenax®-based VOC data, canister-based collection systems have recently been under development. These systems are based on the use of stainless steel canisters which have specially electropolished interiors (McClenny et al., 1986). The proprietary process, known as SUMMA® polishing, passivates the interior walls of the canisters so as to substantially improve the storage capabilities for VOC. Tests have shown that most VOCs of interest can be stored in the canisters for as long as 30 days without significant losses (Oliver et al., 1986). Even reactive species such as ethylene oxide can be kept for at least one week.

The major effort to date has been on pump-based systems which can achieve 24-hour sampling by filling evacuated canisters to 3 atm pressure. A major comparison of the pump-based system with pumped Tenax® samplers (used in the distributed air volume mode: 5, 10, 20 and 40 l; see Walling, 1984); was conducted in a fully furnished, but unoccupied residence in 1986 (Spicer et al., 1986). The HVAC system of the house was spiked with VOCs spanning a range of vapor pressures and chemical types so as to produce concentrations of 3 to 30 mg/m^3 (low ppbv) of these chemicals inside the air of the living space. The agreement between Tenax® distributed air volume (DAV) and canisters was very good. Agreement between the two as characterized by the slope of the linear regression analysis of the Tenax®-collected sample to canister-collected sample was: chloroform, 0.802 + 0.032 (standard error); 1,1,1-trichloroethane, 0.857 + 0.028; tetrachloroethylene, 1.095 + 0.086; bromodichloromethane, 0.862 + 0.018; trichloroethylene 0.916 + 0.019; benzene, 1.081 + 0.141; toluene, 1.030 + 0.056; styrene, 0.928 + 0.036; p-dichlorobenzene, 0.992 + 0.047; hexachlorobutadiene, 0.969 + 0.033 (average of four values). The average ratio was 0.953. This comparison represents an ideal situation in which concentrations are reasonably constant during sampling periods and, for most of the tests, in which temperature and humidity excursions were minimized. The low- and high-rate PSDs were also included in this intercomparison test. The data base resulting from this comparison is currently being examined to determine a number of secondary matters; for example, the changes in agreement when one- or two-tube Tenax® is used. Also of considerable interest are the results of a similar comparisons on ambient air.

A canister-based sampler designed for intermittent sampling over a week-long collection period is being tested to determine the likelihood of component failure. This approach minimizes the need to attend the sampler, at the risk of misrepresenting the contributions of time-varying VOC sources. Typical temporal variabilities for indoor air-related sources are currently being measured. These measurements are possible because of related methods development of continuous sequential samplers utilizing SUMMA®-polished stainless steel syringes. Twelve syringes can be used to sample over periods of as long as eighteen hours and as short as twelve minutes. Commercially available units of this type have been modified to allow interaction between a GC/MS and the sampler by way of GC program commands executed through external electrical latches. This allows automatic transfer of sample from the syringes to the GC preconcentrator followed by an automated analysis. All twelve syringes can be analyzed without manual intervention.

VOC screening procedures for use in reducing the number of canister samples for which detailed GC/MS analysis is done, and for locating likely indoor sources, are also undergoing evaluation. Portable gas chromatographs as well as the devices such as the Photovac TIP that give weighted response to total trace gases in the air can be applied in these cases.

Based on previous extensive work in monitoring personal exposures in TEAM studies and on the generally favorable results obtained in the indoor air intercomparison, single-tube Tenax® and its passive counterpart, the EPA-developed passive sampling device, remain viable alternatives for VOC sampling, at least in situations in which environmental conditions are controlled. Follow-up experimental efforts on the PSDs are in the planning stage. This will include the alteration of an existing commercially available system to accommodate PSDs, Tenax® cartridges, and canisters, so as to facilitate direct comparisons between them. The cause of uncertainties in the sampling rate applicable to the PSDs will also be studied so that further design modifications can be implemented if necessary.

Further research is needed to reduce the costs of the canister-based samplers and to improve the PSDs. New sorbents and mixed-bed sorbent tubes need to be evaluated for improved collection efficiency and reduced artifact problems. Badly needed is a sampling/analytical methodology for small polar organic compounds which are difficult to separate from atmospheric water. These include ethylene oxide and nitriles.

2.6.1.4 Health Effects.

2.6.1.4.1 Neurotoxicity of volatile organic compounds. Monitoring surveys of homes and public buildings worldwide (Berglund et al., 1984) indicate that a bewildering assortment of VOCs is present in such environments. However, the levels of individual VOCs found are generally orders of magnitude below the threshold limit values (TLVs®) that is, levels considered to be harmful to humans exposed for eight hours to any individual compound. Among the VOCs found are acetone, formaldehyde, methylethylketone (MEK), hexane, benzene, toluene, and xylene, used in building materials, furnishings, and adhesives. Chlorinated hydrocarbons frequently found include methylene chloride (paint strippers), trichloroethane (paint), trichloroethylene (type writer correction fluid and degreasing agents) and p-dichlorobenzene (insect repellents). Some of these VOCs (e.g., formaldehyde, benzene) are known to be carcinogenic, as discussed elsewhere in this chapter. Many of the individual VOCs such as n-hexane, MEK, and toluene are also known to be neurotoxic, but at levels much higher than those found in typical new buildings. What is striking, however, in reviewing the known neurotoxicological effects of exposure to many of these compounds, is the commonality of these effects across compounds (Anger and Johnson, 1985). Table 2-35 summarizes neurotoxic effects of some common indoor VOCs. The most frequently observed effects include CNS depression, unconsciousness, vertigo, and visual disorders. Tremor, fatigue, anorexia, weakness, and other neurotoxic effects have been associated with VOCs somewhat less often. Cognitive effects such as memory impairment and mental confusion have also been reported. Neurologic symptoms such as these are recognized in several Scandinavian countries as the "phycho-organic" or "painters" syndrome (World Health Organization, 1985). Although Anger and Johnson (1985) do not indicate the exposure levels at which these effects were observed for individual chemicals, the pattern of documented effects suggests a number of neurobehavioral endpoints that should be evaluated in future indoor air studies. The validity and organic basis of this syndrome are quite controversial (Grasso et al., 1984), but there is extensive literature on the neurobehavioral consequences of occupational exposure to industrial solvents that is germane to the present review. Review of the acute and chronic effects of exposure to industrial solvents, particularly compounds such as "white spirits", should be useful in selecting appropriate measures to assess the neurotoxicity of complex VOC mixtures.

TABLE 2-35. NEUROTOXIC EFFECTS OF VOLATILE ORGANIC COMPOUNDS COMMONLY FOUND IN INDOOR ENVIRONMENTS[1]

Chemical	Neurotoxic Effect[2]														
	A	AN	D	E	F	I	M	MI	P	S	T	U	V	VS	W
Acetone			X									X	X		
Benzene			X	X	X					X		X	X		
*1-Butanol					X							X	X	X	
*2-Butanone (methylethylketone)			X							X		X	X	X	
*n-Butyl acetate			X									X			X
Carbon tetrachloride		X	X		X	X	X						X		
Chloroform	X	X	X	X								X			
Cyclohexane	X		X								X	X	X		
p-Dichlorobenzene										X	X		X	X	X
*1,2 Dichloroethane			X				X	X			X		X	X	
*Ethyl benzene			X										X	X	
Formaldehyde		X		X					X		X			X	X
*n-Hexane	X	X	X					X	X	X		X	X	X	X
Methylene chloride			X		X					X		X			
Styrene		X		X	X			X	X		X	X		X	X
Tetrachloroethylene	X	X	X								X	X			
Toluene			X		X	X	X		X		X	X	X	X	X
1,1,1-Trichloroethane (methyl chloroform)	X	X	X									X			
Trichloroethylene (acetylene trichloride)	X		X		X		X	X			X		X	X	
*3-xylene		X	X	X	X		X	X	X		X	X	X	X	X

*VOCs used in Molhave et al. (1986) mixture

[1] Adapted from Anger and Johnson (1985).

[2] See abbreviation key. Neurotoxic effects listed were generally observed at levels considerably higher than the levels of individual VOCs found in typical indoor environments.

A = anesthesia
An = anorexia
D = CNS depression
E = excitement
F = fatigue
I = incoordination
M = mental confusion
MI = memory impairment

P = paresthesia
S = sensory disturbance
T = tremor
U = unconsciousness, stupor, narcosis
V = vertigo
VS = visual disturbances
W = weakness

Molhave (1985) has hypothesized that the sensory irritant symptoms of sick-building syndrome (SBS) result from the additive or synergistic effects of the complex mixture of VOCs, rather than the specific effect of any individual VOC present in any particular "sick" building. Molhave et al. (1986) have explored the effects of controlled exposure of humans to complex mixtures of VOCs. Results of this study suggest that low-level VOC exposure in amounts comparable to concentrations found in newly constructed Danish homes produced memory impairment and sensory irritation in subjects known to be sensitive to VOCs--that is, persons identified by questionnaire as having "sick building syndrome". These findings are provocative because the available literature on individual compounds contained in the VOC mixture would not indicate any adverse effects at such low levels. The literature concerning the health effects of complex VOC mixtures, however, is negligible. In view of the widespread sensory irritant complaints of workers in new construction and renovated buildings and the known presence of numerous VOCs in such environments, systematic study of the health effects of human exposure to complex VOC mixtures is needed. In particular, the pioneering work of Molhave et al. needs to be replicated and clarified as a first step in exploring the neurotoxicity of VOCs.

2.6.1.4.2 Genotoxicity of volatile organic compounds in relationship to total organic species. Graedel et al. (1986) have recently catalogued, from published sources, compounds found within ambient and indoor environments, their sources, air media (e.g. aerosol, gas,) in which they have been detected and, when available, the genotoxicity test results of these compounds. The chemicals are grouped according to International Union of Pure and Applied Chemists (IUPAC) guidelines. Under the IUPAC chemical classification scheme, a compound is listed only under one class. Table 2-36 provides a condensation of this information for compounds found in indoor air.

Depending upon the source, airborne particle concentration, and humidity, these compounds can be found in the gas phase (e.g., volatile organics), associated with airborne aerosols, or both. Table 2-37 shows that this literature review identified 273 chemicals as indoor air compounds and that 218 (or approximately 80 percent) could be detected as volatile compounds. Of these 218 compounds, 39 were identified as both volatile and aerosol-bound compounds; therefore, 65 percent (179) of identified indoor air compounds were identified only as gases. Of the 273 compounds that have been identified within

TABLE 2-36. PARTITIONING OF INDOOR AIR COMPOUNDS AS INDICATED BY REVIEWED LITERATURE*

Chemical Class	Number Detected	Gas	Gas/ Aerosol	Aerosol
Inorganics	14	8	1	5
Hydrocarbon	110	82	10	18
Ethers	2	1	1	
Alcohols	19	17	1	1
Ketones	10	9	1	
Aldehydes	14	12	2	
Acid derivatives	17	7	1	10
Carboxylic acids	19	1	3	14
O-heterocycles	5	5	1	1
N-organics	10	9	1	
S-organics	2	1		1
Halogenates	38	21	11	6
Organometallics	13	6	7	—
	273	179	39	55

*Extracted from Graedel et al. (1986). Summarized by whether or not each compound was found as a gas or within an aerosol or both (Gas/Aerosol).

indoor air samples, 70 have undergone some degree of genetic toxicology testing (Table 2-37). Among the 70 compounds tested, 31 gave a positive (active) response in at least one bioassay, 33 were negative in the bioassays used, and 6 had a questionable response in at least one bioassay. Twenty-two have been tested in a whole-animal carcinogen bioassay, and 16 of these proved to be carcinogenic in a rodent bioassay. Two chemical classes -- hydrocarbons and halogenated organics -- are worthy of special discussion. Among the 110 hydrocarbons detected within indoor air, only about 10 percent have been tested for mutagenicity or carcinogenicity. Six of these 13 hydrocarbons have been tested in whole-animal carcinogen bioassays, and 4 of these were positive (carcinogenic). Most of these 13 hydrocarbons are formed as a result of some type of combustion process including tobacco smoking, wood combustion, oil combustion, and coal combustion. Some of these hydrocarbons (e.g., toluene, xylene,) are solvents and are associated with adhesives, building materials, printing processes, and paints. For this class of chemicals, short-term bioassays appear to be appropriate screening tests for detecting airborne carcinogens. The other major class of compounds -- halogenated organics, is quite different from the hydrocarbons. Of the 38 halogenated compounds detected within indoor air, 27 have undergone bioassay testing and 13 of these are

TABLE 2-37. CHEMICALS DETECTED IN INDOOR AIR SAMPLING: THEIR OCCURRENCE AND BIOASSAY*

Chemical Class	Number Detected	Number Bioassayed	Positive Bioassay	Positive Animal Carcinogen
Inorganics	14	3	2	0
Hydrocarbon	110	13	5	4
Ethers	2	0	0	0
Alcohols	19	6	1	0
Ketones	10	2	0	0
Aldehydes	14	5	2	0
Acid derivatives	17	3	2	1
Carboxylic acids	19	1	0	2
O-heterocycles	5	1	1	0
N-organics	10	3	2	1
S-organics	2	0	0	1
Halogenates	38	27	13	0
Organometallics	13	6	3	7
				0
Totals	273	70	31	16

*Extracted from Graedel et al. (1986).

positive in some bioassay. Of the nine halogenates tested for animal carcinogenicity, seven were carcinogenic and two gave inconclusive results. These halogenated organics are primarily either solvents (e.g., methylene chloride) or pesticides (e.g., chlordane). One of the halogenated compounds is tetramethyl lead, which is associated almost exclusively with gasoline vapors. Some of the short-term tests, especially bacterial bioassays, are not effective screening systems for the carcinogens that fall into this class of compounds. One must remember, however, that very few gaseous compounds have undergone genetic bioassay testing. Both the identification of aerosol compounds and the testing of volatiles may likely be a function of scientific capabilities and interest rather than a true indication of the distribution of genotoxic compounds between gases and aerosols. Although one recent effort (Claxton, personal communication) has shown that approximately one-half of the total mutagenicity associated with sidestream cigarette smoke is due to the volatile and semivolatile components. Complex mixtures of gases pose special problems for genetic toxicologists. Neither the technology for collecting complex mixtures of gases and returning them to the laboratory for bioassay nor the _in situ_ bioassay methods for testing indoor gases are fully developed. Future efforts are expected to address both of these problems; however, in the meantime individual gaseous compounds will have to be identified and then tested as single components.

2.6.1.5 Mitigation and Control Options.

2.6.1.5.1 Ventilation. The most common technique for reducing the concentration of organic vapors in indoor environments is modifying the ventilation in the affected area (Berglund et al., 1982; Moschandreas et al., 1981b; Billings and Vanderslice, 1982). Increasing the air exchange rate by bringing in outside air will reduce concentration by dilution and flushing. In some cases, this same technique will increase the emission rate by enhancing the vapor pressure driving force for vaporization. Thus, for some compounds higher air exchange rates can reduce concentrations and exposure time. In many cases, however, a simple increase in the amount of dilution air to a building may not be effective. The flow patterns in the affected rooms and the amount of air actually reaching the contaminated zones must also be considered (Skaaret, 1986). Any changes in the ventilation system design or operation will have energy and cost implications that will need to be considered.

2.6.1.5.2 Air cleaners.

2.6.1.5.2.1 Adsorption. A number of systems for collecting organic vapors on adsorbents, primarily activated carbon, are available for indoor air quality control (Research Triangle Institute, 1986). Most are designed for commercial and industrial application, although three manufacturers produce units small enough for residences or single rooms. Units are available both for induct installation or as "stand alone" devices. Almost no data are available on the effectiveness of these units at vapor concentrations below 100 ppm. Adsorbers are also used in submarines to control indoor air quality; both activated carbon and molecular sieves have been employed (Rao, 1986).

2.6.1.5.2.2 Catalytic oxidation. Collins (1986) has reported on a low-temperature catalyst used in a room air filtration device. While designed to remove a variety of indoor contaminants (e.g., combustion gases and tobacco smoke), the device also reduced the concentration of acetaldehyde, acetone, methylene chloride, and MEK. Benzene was not effectively removed. Rao (1986) reports on the use of catalytic oxidation for cleaning the air in submarines. Further evaluations are required to determine the effectiveness of catalysts in reducing the concentration of organic vapors in indoor environments.

2.6.1.5.3 Material/product selection. The levels of organic vapors in the indoor environment can be affected by the selection of materials and products. As discussed previously, HUD limits the emission rates of formaldehyde from pressed wood products used in mobile homes to reduce the concentrations in these residences. Urea formaldehyde foam insulation (UFFI) is no longer used due to excessive emissions. Denmark requires the labeling of epoxy and polyurethane products to inform the consumer of potential health hazards (Andersen et al., 1982). Judicious selection of low-emission materials and avoidance of known irritants would limit exposures. Experiments by Molhave et al. (1984) showed that, for a typical mixture of organic compounds found in indoor air, people start to become irritated at a total concentration of about 5000 $\mu g/m^3$. Since a typical building has many sources that contribute to the total concentration, Tucker (1986a,b) has suggested that it would be prudent to keep the contribution of any one source below about 1000 $\mu g/m^3$. In very rough terms this means that material sources should be avoided or conditioned if they emit more than 100 mg/hr of organic compounds per 100 m^2 of residential space with mixed and well distributed air, or sources emitting more than 400 mg/hr per 100 m^2 of typical office space with well mixed and distributed air. Unfortunately, most indoor products have not been sufficiently characterized to allow such selection to occur.

2.6.1.5.4 Material/product use. The manner in which products are used can impact indoor organic vapor concentrations. Emissions of formaldehyde from pressed wood products decline with time (Hawthorne et al., 1984), so aging or conditioning of such products prior to installation would reduce emissions. Solvent-containing materials (e.g., paints, adhesives, caulks, paint removers, and waxes) should be used in well ventilated areas. Manufacturers' instructions should be followed. Some activities, such as hobbies, woodworking, and paint stripping, which use high-emission products should be isolated and separate exhaust fans provided. Solvents and solvent-containing products should be stored in airtight containers; outside storage is preferred.

2.6.1.5.5 Other measures. Other measures for controlling the levels of indoor organic vapors include:

- building "bake out", the process by which new or renovated buildings are heated and ventilated prior to occupancy (State of California, 1984); and

- formaldehyde removal via fumigation. Dement et al. (1984) report that fumigation with 1000 ppm of ammonia for 24 hours reduced formaldehyde emissions by more than 70 percent. The long-term effectiveness of this approach is not known.

2.6.2 Formaldehyde

One noncombustion gas-phase organic compound is HCHO, which is also a product of combustion. This is a colorless, pungent gas which is highly water soluble and hence irritating to the mucous membranes of the eyes and respiratory tract. It is classed as a VOC, but because of the prevalence of its use in building materials such as particleboard, carpeting, and cloth finishing, it is described separately here.

2.6.2.1 Sources of Formaldehyde. Formaldehyde is emitted from UFFI and from resins used in plywood and particleboard. Emissions from gas stoves and burning cigarettes also contain formaldehyde (Repace, 1982). Carpeting may also be a source of formaldehyde (Molhave, 1982; Pickrell et al., 1984). Anderson and Lundquist (Pepys, 1982) concluded that the use of particleboard and plywood in furniture construction causes inordinately high levels of formaldehyde due to the use of urea-formaldehyde glue in construction. Many textile products such as draperies, rugs, and upholstery fabrics may have a long-lasting emission of formaldehyde due to the treatment the textiles receive.

Most of the material source research done to date in this country has been on formaldehyde emissions from pressed wood products, especially the various types of particleboard. Regulations for formaldehyde emissions from pressed wood products were established by HUD in 1984. Prior to that time, research measurements at Oak Ridge National Laboratory (ORNL) and other laboratories were being made so as to understand how ventilation parameters like temperature, relative humidity, and air exchange rates affect emission rates of formaldehyde. Although such work continues at ORNL, the National Bureau of Standards (NBS), and Georgia Tech Research Institute, formaldehyde emission measurements have shifted more toward testing of products to determine compliance with the HUD standard.

2.6.2.2 Monitoring of Formaldehyde. The lack of a sensitive, accurate and inexpensive method for monitoring formaldehyde in nonoccupational environments has been a serious obstacle to the performance of exposure studies and has prompted the development of improved techniques for the collection and analysis of formaldehyde.

Three PSDs and two active sampling devices have been considered by EPA as PEMs for formaldehyde. The PSD which has seen the most use, especially in Canada, is a dry sulfite device manufactured by Air Quality Research, Inc. (AQR) (Geisling et al., 1982). The AQR PSD (PF-1) resembles a Palmes tube and is designed for indoor area monitoring; however, it could be worn on the person. It has a sensitivity of about 2 ppm-hr; therefore, five to seven days of exposure are generally necessary for nonoccupational levels of formaldehyde. In 1985 EPA conducted chamber tests of the AQR PF-1 device and compared it directly with an active sampler (Snow, 1985). It was recommended that the PF-1 not be used for exposure periods of less than four days because sampling rates were found to be time-dependent.

A new commercial PSD from the Air Technology Labs., Inc. (ATL) is the passive bubbler monitor. The ATL device employs a permeable membrane to separate the reagent (3-methyl-2-benzothiazolone hydrazone, or MBTH) from the atmosphere sampled. The manufacturer claims a detection limit as low as 0.1 ppm for a 2-hr exposure, and the device has a distinct advantage in that it can be developed and read by untrained personnel in the field. To date, however, EPA has not had funds to test this device.

Prior to the advent of the ATL PSD, a Canadian company known as Crystal Diagnostic, Inc. (CDI) introduced a prototype PSD containing a film of monodispersed hydrobenzoic acid hydrazide. Upon exposure to formaldehyde, crystal nuclei form, which after development can be read visually by an untrained individual in the field. Attempts to improve the sensitivity of the CDI device to permit detection of 10 ppb of formaldehyde after 8- to 24-hr exposures have not been successful. CDI has recently been acquired by Foxboro, which stated that it intends to continue developing the PSD.

There are two pumped-tube methods for measuring formaldehyde which do have adequate sensitivity and can be used for either indoor air or personal exposure monitoring. Both utilize solid sorbents coated with 2,4-dinitrophenylhydrazine (DNPH) and require analysis by HPLC. Consequently, analytical costs are high. One method developed by Lipari of General Motors Research Laboratory (Lipari and Swarin, 1985) is applicable only to formaldehyde and has a detection limit of less than 2 ppb hr. The other method, developed by EPA (Tejada, 1986), has comparable sensitivity but offers the added advantage of the capability to simultaneously determine other aldehydes and ketones. In 1986 these methods were compared by EPA for outdoor air monitoring and found to have problems with blanks used to zero the monitors for measurement of samples. These problems should, however, be minimal for indoor applications where formaldehyde concentrations are generally much higher than for ambient air. It is anticipated that the blank problems can be eliminated through better packaging for shipment to and from the laboratory. EPA has selected the Tejada method (Tejada, 1986) for inclusion in the Toxics Air Monitoring System (TAMS) in 1987.

2.6.2.3 Health Effects. Because of formaldehyde's high water solubility, it causes irritation to the mucous membranes of the eyes and upper respiratory tract. A variety of short-term signs and symptoms are commonly accepted as associated with formaldehyde exposure; some occur at levels that have been measured in residential air (Table 2-38) (National Research Council, 1981a). However, other acute and chronic health effects of formaldehyde are more controversial, including human carcinogenicity, effects on the lungs, and neurobehavioral impairment.

TABLE 2-38. ACUTE HUMAN HEALTH EFFECTS OF FORMALDEHYDE AT VARIOUS CONCENTRATIONS*

Formaldehyde Concentration (ppm)	Reported Effects
0.0 - 0.5	None reported
0.05 - 1.5	Neurophysiologic effects
0.05 - 1.0	Odor threshold
0.01 - 2.0	Eye irritation
0.10 - 25	Upper airway irritation
5 - 30	Lower airway and pulmonary effects
50 - 100	Pulmonary edema, inflammation, pneumonia
> 100	Death

*Adapted from Table 7-2 (National Research Council, 1981a).

As measured by determination of optical chronaxy, electroencephalography, and sensitivity of dark adapted eyes to light.

The low concentration (0.01 ppm) was observed in the presence of other pollutants that may have been acting synergistically.

Formaldehyde is primarily deposited in the upper respiratory tract, including the nasopharynx; and cancer of structures in this region could be a result of formaldehyde exposure. In 1979, it was reported that rats exposed to formaldehyde developed nasal cancer, a tumor rarely found in unexposed animals

(Kerns et al., 1983). This report, subsequently confirmed (Albert et al., 1982), stimulated the rapid performance of a number of epidemiological investigations.

Occupational exposure to formaldehyde has been examined as a risk factor for nasal cancer, as well as for several other sites, in both case-control and cohort studies. Retrospective cohort studies of formaldehyde-exposed workers have not shown excess nasal cancer, but their statistical power has been limited (Blair et al., 1986; Stayner et al., 1986; Acheson et al., 1984; Levine et al., 1984; Marsh, 1983). Some case-control studies have shown associations between measures of formaldehyde exposure and nasal cancer (Olsen et al., 1984; Hayes, 1986), whereas others have not (Brinton et al., 1985; Hernberg et al., 1983). The lung, the buccal cavity, and the pharynx have also been examined as sites for formaldehyde-related malignancy (Blair et al., 1986; Stayner et al., 1986; Acheson et al., 1984; Harrington and Oakes, 1984; Levine et al., 1984).

Based on the results of occupationally and domestically exposed populations, formaldehyde has been reported to cause excessive respiratory symptoms, acute and chronic reductions of lung function, and asthma. Questionnaire surveys on symptoms have been carried out in populations selected because of complaints about formaldehyde exposure from UFFI (Table 2-39). These surveys show high symptom prevalences, but their results may have been biased by the selection of subjects with complaints. A more informative and potentially less biased design was used in a study of residents of homes insulated with UFFI and of nonexposed controls (Thun et al., 1982). During the year before interview, the prevalence of wheezing and burning skin was significantly higher for residents of homes with UFFI. Subjects who reported that odor had persisted for longer than seven days after installation of UFFI had the highest incidence of symptoms. Excessive respiratory symptoms have also been found in workers exposed to formaldehyde in mobile homes used as offices (Olsen and Dossing, 1982; Main, 1983).

Formaldehyde has been shown to be a cause of occupational asthma, although its mechanism of action is uncertain (Hendrick and Lane, 1977; Imbus, 1985). Studies of individuals exposed to formaldehyde in their homes have documented complaints of wheezing, chest tightness, and other symptoms compatible with asthma (Table 2-40). However, cases of asthma resulting from domestic exposure to formaldehyde have not been published. In a documented case of a woman who

TABLE 2-39. SURVEYS OF OCCUPANTS LIVING OR WORKING IN MOBILE HOMES OR HOMES WITH UFFI

Study Population	Findings	Comments
424 adults, 99 children living in 334 mobile homes. Complaint* investigations, Washington State (Breysse, 1979).	Adults (A); Children (C) Eye irritation: A - 58(%) C - 41 Throat irritation: A - 66 C - 62 Chronic headache: A - 40 C - 16 Chronic cough: A - 9 C - 33 Memory lapse/ drowsiness: A - 24 C - 7	Formaldehyde levels: 0.03 - 1.77 ppm; No control group; Exposure-response not examined
256 adults and children living in 65 mobile homes or 35 other structures Complaint* investigations, Wisconsin (Dally et al., 1981).	Eye irritation: 68(%) Throat irritation: 57 Headache: 53 Cough: 51 Difficulty sleeping: 38 Wheezing: 20	Formaldehyde levels: 0.0 - 3.68 ppm; No control group; Exposure-response not examined
162 residents of 68 homes with UFFI. Complaint* investigations Connecticut (Sardinas, 1979).	Eye irritation: 39(%) Nose/throat/lung irritation: 48 Headache: 17 No apparent relationship between symptoms and crude formaldehyde level	Formaldehyde levels: 0.0 - 10 μg/l, with detectable and nondetectable levels
Unknown number of residents in 443 families living in mobile homes. Complaint* investigations, Texas (Norsted et al., 1985).	No difference in symptom prevalence in families living in homes with and without detectable levels	Formaldehyde levels: 0.0 - 8 ppm. Comparison of homes with detectable and nondetectable levels
1396 residents of UFFI homes; 1395 residents of non-UFFI homes. Retrospective cohort, New Jersey (Thun et al., 1982).	Exposed more likely to report wheezing than nonexposed: Wheezing: Exposed - 0.6(%) Nonexposed - 0.1 Burning skin: Exposed - 0.7 Nonexposed - 0.1 Subgroup, in whose homes odor persisted >7 days after foam installed, had higher symptom incidence	Population-based study. Formaldehyde concentrations not measured

(continued on following page)

TABLE 2-39. (continued)

Study Population	Findings	Comments
70 exposed employees of 7 mobile home care centers, 34 nonexposed employees of 3 permanent structures, Denmark (Olsen and Dossing, 1982).	Exposed reported significantly more symptoms than nonexposed: Menstrual irregularities Exposed - 35(%) Nonexposed - 0 Excessive thirst: Exposed - 60 Nonexposed - 5 Eye irritation: Exposed - 55 Nonexposed - 15 Headache: Exposed - 80 Nonexposed - 50	Formaldehyde levels in mobile day care centers: 0.24 - 0.55 ppm; permanent structures: 0.05 - 0.11 ppm
21 exposed workers in mobile home office, 18 nonexposed workers in another office, Illinois (Main, 1983).	Exposed reported significantly more symptoms: Eye irritation: Exposed - 81(%) Nonexposed - 17 Throat irritation: Exposed - 57 Nonexposed - 22 Fatigue: Exposed - 81 Nonexposed - 22 Headache: Exposed - 76 Nonexposed - 11 No difference in pulmonary function	Formaldehyde levels in offices ranged from 0.12 - 1.6 ppm

*Complaint investigations were initiated at residents' requests.

developed asthma after installation of UFFI, the offending agent was found to be UFFI dust rather than formaldehyde (Frigas et al., 1981). Frigas et al. (1984) evaluated 13 subjects referred for evaluation of possible asthma secondary to formaldehyde exposure in the work or home environment. None of the 13 subjects responded to formaldehyde challenge. There is concern, however, that it may cause severe allergenic response to as much as 8 percent of the population.

Surveys of symptoms in subjects concerned about formaldehyde exposure in their homes have shown a high prevalence of such neuropsychological symptoms as in headache, memory lapse, fatigue, and difficulty in sleeping (Breysse, 1979;

TABLE 2-40. STUDIES OF FORMALDEHYDE EXPOSED COHORTS AND CANCER

Study	Findings	Comments
Cohort study of pathologists, Great Britain (Harrington and Shannon, 1975)	SMRs elevated for lymphoma and hematopoietic neoplasms (211) but not for leukemia	Less than 10% of cohort deceased. Less than 20 yrs of follow-up
Proportional mortality study of embalmers, New York (Walrath, 1981)	PMRs significantly elevated for cancers of skin (221) and colon (143); nonsignificantly for cancers of brain (156) and kidney (150), and leukemia (140)	
Proportional mortality study of embalmers, California (Walrath and Fraumeni, 1983)	PMRs significantly elevated for cancers of colon (188), brain (191), and prostate (176), and leukemia (174); nonsignificantly for bladder cancer (138)	
Cohort study of pathologists, Great Britain (Harrington and Oakes, 1984)	SMRs significantly elevated for brain cancer (300) but not lymphoma	Less than 5% of cohort deceased. Six yrs of follow-up
Cohort study of anatomists, U.S.A. (Stroup, 1984)	SMRs elevated for brain cancer 271, 95% (CI = 130-499) and leukemia (148, 95% CI = 71-272)	Excess brain cancer persisted when psychiatrists used as a reference group
Cohort study of undertakers, Canada (Levin et al., 1984)	SMRs nonsignificantly elevated for brain cancer (115) and leukemia (160)	20 yrs of follow-up
Proportional mortality study of chemical plant employees, Massachusetts (Marsh, 1982)	PMR nonsignificantly elevated for cancers of digestive organs (152) among formaldehyde exposed workers. No data reported on brain cancer and leukemia	No evidence of trend of mortality in relation to exposure
Cohort study of chemical plant employees, U.S.A. (Marsh, 1983)	SMR significantly elevated for cancers of genitourinary tract (169). SMR for leukemia not elevated. No data for brain cancer	Case-control study within cohort showed no association between GU cancer and a general plant exposure
Cohort study of chemical plant employees, Great Britain (Acheson et al., 1984)	SMRs for lung cancer significantly elevated (124) in one of six men most highly exposed	Retrospective assessment made of level of exposure

SMR = Standard Mortality Ratio

PMR = Proportional Mortality Ratio

CI = Cancer Incidence

GU = Genitourinary

Dally et al., 1981; Sardinas et al., 1979). Formaldehyde exposures in these uncontrolled studies ranged up to about 4 ppm. Schenker et al. (1982) used standardized respiratory and neuropsychological tests to evaluate 24 residents of homes insulated with UFFI. Complaints of memory loss were not validated by formal tests but 11 of the 14 subjects who were tested had a deficit of attention, and 9 of the 11 had elevated depression scores. Two controlled studies of the neuropsychological effects of formaldehyde exposure in the occupational setting also suggest similar effects (Olsen, 1982; Kilburn, 1985).

2.7 RADON

2.7.1 Occurrence and Sources of Radon

Radon-222 (hereafter called radon) is a noble gas with an atomic weight of 222, which makes it the heaviest known gas. It cannot be detected by the human senses and it is relatively soluble in water at room temperature. It is radioactive, emitting alpha particles, with a half-life of 3.8 days (Evans, 1969). Radon gas is a decay product of radium-226, which is itself a decay product of the uranium-238 series. The first four radon decay products (RDP), polonium-218, lead-214, bismuth-214, and polonium-214, are relatively short-lived, each having a half-life of less than 30 minutes. Radon, polonium-218, and polonium-214 are alpha emitters, whereas lead-214 and bismuth-214 are beta and gamma emitters. Polonium-214 decays to lead-210 with a half-life of 22 years, which effectively terminates the series as far as lung deposition is concerned. This decay series actually ends with the stable, nonradioactive element lead-206.

Trace amounts of naturally radioactive nuclides are present in soil, rock, and building materials, as well as in living organisms, including human beings. Radon and its short-lived decay products in indoor air are the greatest contributors to the total dose burden from natural radioactivity. In particular, it is the alpha-emitting polonium-218 and polonium-214 decay products or radon daughters that are of most concern (National Council on Radiation Protection and Measurements, 1984a,b).

Because of the relatively short half-life of the alpha-emitting radon daughters (<30 min, in aggregate), significant concentrations cannot be maintained without a radon source. The daughters will reach equilibrium with radon rapidly when no separative or removal processes are active. This permits radon concentration and its variability to serve as an indicator of possible radiation dose to the lung by short-lived daughters. For dose calculation however, the alpha particles emitted by both radon and its progeny at equilibrium must be considered.

Overall estimates of global emanation of radon identify soil as the greatest contributor of radon, followed by ground waters and the oceans. Atmospheric concentrations of radon are estimated to average between 100 to 200 pCi/m^3 (3.7 to 7.4 Bq/m^3), with concentrations over uranium ore-grade soil typically between 500 to 1000 pCi/m^3 (18.5 to 37 Bq/m^3).

Because uranium-238 and, consequently, radium-226 exist to some extent in most soils and rocks, radon is also widely distributed in these materials. Because radon is a gas, it has the potential to diffuse through pores and cracks in soil and rock to mix with other soil gases and to escape into the atmosphere. The short-lived RDPs are very small particles which agglomerate rapidly and readily attach to surfaces, including suspended particles or water droplets in air. Radon may enter a dwelling directly with the soil gas, in solution with the water, or by diffusing from construction materials.

Nazaroff (1984) suggests that the infiltration of soil gas directly into single-family homes is the largest contributor to indoor radon levels. Radon formed from radium in the soil or rock diffuses into the interstitial spaces and migrates to the surface with the soil gas. Soil with coarse grain size, gravel, and sand are highly permeable and allow more migration than muds and clays which have finer grain sizes and higher moisture contents. Radon reaching the soil surface is diluted in the air. However, it may reach a surface that is in contact with a structure such as a basement wall or floor or a slab-on-grade foundation. If pathways such as cracks and holes are present in the structure, and the air pressure is lower in the structure than in the soil, radon flows into the structure. Higher than normal concentrations of radon in the soil or higher than normal permeability of the soil surrounding a dwelling can contribute to increased rates of radon entry. The combination of soil permeability and pressure difference between the soil gas and the air inside the structure largely determine the effective volume of radon that enters the structure.

Indoor levels of radon have been measured to range from the lower limit of detection 500 pCi/m^3 to 2 x 10^6 pCi/m^3 (ca. 18 to 7 x 10^4 Bq/m^3). This variability of three orders of magnitude of the radon concentration indoors should be considered the working range for commonly needed indoor radon measurements, although a variability of less than one order of magnitude is expected on a house-specific basis. Radon daughters in equilibrium with these levels of radon should vary in the same manner. Equilibrium ratios of the activity concentration of radon to radon progeny in indoor environments vary from near 1.0 to 0.3 at higher indoor ventilation rates.

While there is consensus in the radon/radon progeny research area that radon progeny in indoor environments pose one of the greatest environmental risks, there is scientific debate as to what indoor conditions and ambient air characteristics are most closely related to the observed health effects. This led the National Council on Radiation Protection and Measurements to recommend "that future (radon and radon progeny) measurements should provide the aerosol data to allow calculation of the specific bronchial dose as well as documenting exposure . . ."(National Council on Radiation Protection and Measurements, 1984a,b). In particular, this was a call for determination of the aerosol size and number to which radon progeny would attach themselves, as well as a determination of the partitioning between attached and unattached radon progeny under various aerosol conditions.

Nero (1985) notes that homes served by private wells in areas with high soil radium content have a high probability of increased radon levels in the tap water. Water with elevated radium levels has correspondingly high radon levels, groundwater being more likely to have higher levels than surface water. Radon can also be dissolved directly in the water as it moves through the soil. Approximately 50 percent of U.S. homes are served by private wells, lakes, and streams (Smolen, 1984; U.S. Environmental Protection Agency, 1985b). The radon is released from the water at elevated temperatures, and when the water is highly agitated, or in finely divided droplets with large aggregate surface area, such as occurs in a shower, a dishwasher, or a washing machine.

Certain technological activities are known to cause elevated levels of indoor radon in some locations. Phosphate industry waste materials in Florida and uranium mill tailings in Colorado, both of which contain elevated concentrations of radium-226, have resulted in elevated indoor radon levels when these products were placed under and around structures. Waste from a radium processing facility in New Jersey was also observed to contribute to elevated radon levels in some structures.

Radon is also released from many building materials, but normally at very low levels. Wood materials tend to emanate the least radon, while brick, cement, and cinder block emanate more. Radon is released from all of these sources at such a low rate that rarely are these materials an important contributor to elevated radon levels. However, there have been a few cases in which materials containing significant radium concentrations were used to form building materials. Examples of these situations are houses that have been built with materials contaminated with uranium or radium mill tailings and uraniferous phosphogypsum waste utilized as drywall. Another example is homes that use radium-containing heat storage rocks (e.g., large pieces of granite) and circulate large volumes of air into the living areas.

2.7.2 Indoor Concentrations and Exposures

The detection of radon and RDP in air depends upon the interactions of alpha and gamma radiation from the decay products with some detection media. For example, alpha particles may interact with zinc sulfide to produce light pulses, which are counted using a photomultiplier tube assembly. Alternatively, alpha particles interact within a polycarbonate plastic chip to produce ionization tracks, which are enlarged through etching and then visually counted. Gamma radiation from decay products collected on activated carbon canisters may interact with sodium iodide crystals to produce light pulses, which are counted. Other interactions and detection schemes exist and are used in a variety of instruments and methods depending on the levels to be measured, the accuracy required, the equipment available, and the convenience of the operator.

Instrumentation is available to analyze indoor air samples for radon and radon progeny (with acceptable accuracy and precision). EPA guidance has been published for the use of continuous monitors (scintillation cell), 3- to 5-day integrating charcoal canisters, alpha-track detectors, and grab sample scintillation cells (U.S. Environmental Protection Agency, 1986). This instrumentation has been and is being employed in various local and regional air quality monitoring and EPA radon mitigation studies. Because of the private sector involvement in radon mitigation activities and the need to verify reductions in indoor radon levels, the EPA Office of Radiation Programs (ORP) has maintained a measurement proficiency program to ensure the quality of both private and public sector measurement programs.

A passive radon badge (Terradex Track-Etch) is also available and has suitable accuracy for monitoring most indoor microenvironments. However, exposure times of several months are usually required for measurable change to occur.

Radioactivity is measured in terms of the rate of decay of a particular unstable nuclide. For instance, the Curie (Ci) is defined to represent 3.7×10^{10} disintegrations per second. Radon concentrations are usually measured in pCi/l (picoCuries per liter), where a picoCurie is 10^{-12} Curies. For occupational historical reasons, the concentration of RDP is generally expressed in working levels (WL), where one WL is any combination of RDP in one liter of air that ultimately releases 1.3×10^5MeV (million electron volts) of alpha energy during decay (Holaday et al., 1957). A concentration of 1 pCi/l of radon in dynamic equilibrium with its decay products, translates into about 0.005 WL. Since exposure depends upon both concentration and time, the occupational historical radon exposure unit has been the Working Level Month (WLM). The WLM was developed to describe exposure sustained during the average number of hours spent underground by miners, where 1 WLM represents the exposure to 1 WL for 170 hours. The relationship between exposure, measured in WLM, and dose to the tissues of the lungs, measured in rems (the traditional biological units) is quite complex and will not be elaborated upon here.

Nearly all structures have some indoor radon which is expected to be at least as great as that found outdoors; however, concentrations above these normal levels arise when the above-mentioned sources are present. Naturally occurring radon in the outdoor air ranges from 0.1 to 1 pCi/l, where 1 pCi/l corresponds to 6.5×10^{-9} $\mu g/m^3$ of radon gas. Note that this concentration is 10 orders-of-magnitude smaller than the proposed new ambient air quality standards for particulates (PM-10 standard). This example serves to illustrate the extremely small concentrations of radon which are environmentally significant. For instance, EPA recommends remedial action for indoor concentrations of radon which exceed 4 pCi/l (2.6×10^{-8} $\mu g/m^3$).

A few areas in the United States are known to have geological factors that contribute to elevated indoor radon concentrations. These factors include above-normal concentrations of radium in the soil and the presence of porous soil or fractured rock formations affording ready migration of radon into structures. This combination of conditions exists in the Reading Prong, a geological area located in parts of Pennsylvania, New Jersey, and New York, where extremely high indoor radon levels have been observed. Indoor radon levels 1000 times the recommended action level have been observed in a few instances. In these cases, the observed concentrations of radon in the soil gas were very high.

Most deep water supplies in the United States have radon concentrations of less than 2000 pCi/l. Some areas, however, such as Maine, have levels exceeding 100,000 pCi/l (Walsh et al., 1984). On the average, it has been estimated that drinking water contributes only 2.5 percent to the background radiation dose (Cothern, 1986).

The average amount of radon in public water supplies is less than 500 pCi/l and does not pose usually very great risks. Whether or not radon and its progeny leave the water depends on the temperature and surface area of the water. If the water is near boiling, in finely divided droplets, and agitated as in a dishwasher, up to 90 percent of the radon contained in the water can enter the air. Even in showers, a considerable portion may leave the water, attach to water vapor, and be inhaled.

There is a need to determine indoor activity concentrations of radon and radon progeny over a wide range of concentrations for multiple purposes; for example, for health risks assessments, determining the location and variability of the radon sources, and determining the effectiveness of indoor radon control techniques. For this purpose field-hardy instrumentation is needed to determine aerosol concentration and size distributions and the partition of attached and unattached radon progeny under various aerosol conditions resulting from ventilation or the operation of air cleaning devices. A small passive device for detecting radon progeny is needed, as well as instruments for calibration of radon/radon progeny measuring devices.

2.7.3 Health Effects Associated with Radon Exposure

The only documented health effect associated with exposure to high concentrations of radon is the increased risk of lung cancer. Alpha-emission from inhaled RDPs in the respiratory tract is thought to produce the tissue injury that eventually results in malignancy. The alpha particles are presumed to penetrate into the airway epithelium and damage the genetic material of the basal cells. The RDPs also release low-energy gamma and beta particles during decay, but the damage to the lung is almost exclusively due to the alpha particles released by polonium-218 and polonium-214 (National Council on Radiation Protection and Measurements, 1984a,b).

The primary concern when discussing the risks from exposure to radon is not the exposure to the radon gas itself, but exposure to its decay products, namely the two alpha-emitting isotopes of polonium. Under normal circumstances, the RDP rapidly attach to airborne dust particles. The RDP that do not attach to aerosol particles agglomerate readily to form very small particles in the size range from about 0.002 μ to perhaps as large as 0.02 μ. These agglomerates are referred to as the unattached RDP or unattached radon progeny. The size of the particles to which the RDP is attached is very important in determining the location in the respiratory system that will receive the radiation dose. Particles readily stick the the moist epithelial lining of the bronchi. Most dust particles are eventually cleared from the bronchi by mucous, but not quickly enough to keep the bronchial epithelium from being exposed to alpha radiation from the decay of polonium-218 and polonium-214. The alpha particles that penetrate the epithelium cells can deposit sufficient energy in the cell to kill or transform it. The transformed cell has the potential to induce a lung cancer. Since lung dosimetry models have indicated that the calculated dose to the lungs from unattached decay products may be greater than from attached decay products, there may be a significant health risk distinction between these two cases (James, 1984; Sextro et al., 1984). The health effects associated with the inhalation of RDPs depend on where in the lung the particle is deposited. In the upper portion of the respiratory system, particles are continuously cleared by the ciliated mucous lining. However, particles deposited deep in the unciliated bronchiolar or alveolar regions of the lung have long residence times and, consequently, may have greater adverse health effects. The larger particles have a higher probability of depositing in the upper regions of the lung, whereas only the small particles, such as the unattached decay products, have a high probability of being deposited deep in the lungs. Therefore, the mathematical models predict a higher lung dose associated with unattached RDPs. One implication of this conclusion is that the use of an air cleaner to remove particles from the air may increase the fraction of unattached decay products and, consequently, increase the radiation dose to the lungs. The results of these predictions have not yet been confirmed by measurements, therefore, the effects of using air cleaners to reduce the health risks from RDP are not established.

2.7.4 Risk Estimates

The lung cancer risk associated with exposure to RDP was first recognized in underground miners. While the lung cancer risk incurred by underground miners has been recognized for a century, the hazards posed by environmental RDPs have only recently become a public health concern. Numerous studies of uranium miners and other underground miners have established a causal relationship between exposure to RDP and lung cancer (Lundin et al., 1971; National Research Council, 1980). Animal studies confirmed that exposure to RDP alone causes lung cancer (National Council on Radiation Protection and Measurements, 1984a). The human data come primarily from miners having high exposures, and the risks of lower exposure levels have not yet been well characterized. Risks estimates associated with the low exposures typical of low indoor radon concentrations were obtained by extrapolations and, consequently, are somewhat uncertain. These uncertainties may soon be removed through the analysis of more recent data on groups of miners whose exposure levels lie within the range of typical residential exposures (Muller et al., 1983; National Institute of Occupational Safety and Health, 1985; Solli et al., 1985; Howe et. al., 1986). It is also true that data is currently being collected in which residential exposures were much higher than any from underground mines. These data should provide good cross checks for risk calculations.

The U.S. Environmental Protection Agency (1985a) estimated that the aggregate health effects over all radon exposure levels range from about 5000 to 20,000 lung cancer deaths per year. The dose to the bronchial epithelium of the lung is thought to come primarily from the unattached radon daughters (Shapiro, 1956; National Council on Radiation Protection and Measurements, 1984a,b).

2.7.5 Estimate of Dosage to People Exposed to Radon

Estimating a person's incurred dosage of radiation produced by a measured concentration of radon in a residence is difficult. Measuring the physical presence of radon in a volume of air is relatively simple; its concentration is usually reported in terms of associated disintegrations per second. Quantifying the amount and significance of the radiation that actually reaches various body tissues is, however, dependent on several parameters that vary and interact in complex ways. The link between a given air concentration of radon and the consequent radiation dosage involves the following principal factors, about which there are varying opinions and degrees of consensus:

- ratio of indoor radon levels (primarily from soil seepage and emanations from building materials) to outdoor, background levels;
- presence of radioactive precursors (most important in mining and industrial settings);
- presence of 'radon daughters', the products of further radioactive decay, and their contributing effects;
- proportion of time spend in the indoor environment;
- breathing rate (activity level) which determines the amount of contaminated air brought into direct contact with susceptible lung tissue cells;
- retention rate in the lung of the 'radon daughters'.

W. F. Bale (1980) equates a radon concentration of 1 x 10^{-10} Curies per liter (Ci/l) with an accumulated dose of 22.6 rem (roentgen equivalent man) for a 40-hr work week. Expressed per 100 picoCuries, this is:

$$100 \text{ pCi/l} = 22.6/40 = 0.565 \text{ rem/hr.}$$

Another source, Snihs (1985), equates a radon concentration of 200 Bequerel/m^3 (Bq/m^3) with a yearly dose of 20 milliSieverts (mSv). From Burkart (1986), 200 Bq/m^3 can be equated with 5.4 pCi/l and 20 mSv/yr with 200 mrem/yr. Thus, 100 pCi/l = 3704 mrem/yr. Assuming 75 percent occupancy (6570 hrs/yr), this becomes:

$$100 \text{ pCi/l} = 0.564 \text{ mrem/hr.}$$

For purposes of estimating dose here, a radon concentration of 100 pCi/l will be equated with a dose of 0.5 mrem/hr.

2.7.6 Indoor Air Quality Control Options

Basically, the two options available for controlling the concentration levels for indoor radon are: 1) removing it once it has entered the structure, or 2) preventing it from entering the structure. The removal method that has been demonstrated to be most effective in reducing indoor radon levels is forced ventilation. Natural ventilation consists of the exchange of indoor and outdoor air in response to natural driving forces. The major sources of the natural driving forces are winds, and temperature and pressure differences between indoors and outdoors (Perdue et al., 1980). With windows and doors closed, typical American homes are characterized by a natural annual average ventilation rate of about 1 ach (air changes per hour). Energy efficient homes may have exchange rates as low as 0.1 ach whereas older homes may have rates as high as 2 ach. Ventilation is effective in reducing the concentration of indoor radon by replacing a certain volume of contaminated indoor air with an equal volume of relatively uncontaminated outdoor air which then proceeds to further dilute the remainder of the indoor air. By simple dilution, increasing ventilation rates over the range from 0.25 to 2.0 ach can yield up to 90 percent reduction in indoor radon concentrations. Even at very high exchange rates, ventilation will not reduce radon levels below some specific value which depends on the radon entry rate. Since the absolute effectiveness of ventilation to reduce radon levels decreases with increasing ventilation rate, this method of reduction will be most cost-effective for tight (low air exchange rate) homes.

A more cost-effective method for removing indoor radon uses forced ventilation with heat recovery. Heat recovery is usually accomplished through an air-to-air heat exchanger. These are low-pressure drop devices that exchange heat from the warmer air being exhausted to the incoming cooler air (or vice versa if an air conditioner is in operation). These devices are sometimes equipped with auxiliary heaters to compensate for the remainder of the heat loss. Air-to-air heat exchangers operate with nominally balanced supply and exhaust flows so the pressure in the building is unchanged. Consequently, both the natural ventilation and soil gas flows are unchanged, which means the radon concentration in the building is affected only by the ventilation rate. At best, radon levels can be reduced by a factor of two or three using forced ventilation with heat recovery (Nazaroff et al., 1981). If the ventilation system were operated in a manner which leads to a depressurization of the house, the radon level might actually be increased.

Although there are several methods for reducing the concentrations of indoor radon and its decay products, most investigators agree that the most practical and desirable approach is to prevent its entry in the first place (Nero, 1985; U.S. Environmental Protection Agency, 1986c). Four general approaches that will prevent radon entry from the common sources are: 1) sealing entry points, 2) ensuring that the direction of airflow is from the house into the soil, 3) removing radon from the water supply, and 4) avoiding the use of building materials that contain significant quantities of radium.

In most instances, the major source of indoor radon is soil gas entering the house. Consequently, a major first step in preventing the entry of radon is to seal the major entry points such as exposed earth in basement floors or drainage sumps. Several studies have shown that sealing all visible cracks and gaps between floor, walls, and service pipes can significantly reduce radon concentrations in houses with radon levels in the range of 30 to 70 pCi per liter (New York State Energy Research and Development Authority, 1985; Holub et al., 1985). A word of caution concerning sealants is in order. Because radon entry is usually pressure gradient driven, very small cracks or openings can provide effective pathways. Therefore, very slight movements of the house substructure may reopen sealed pathways. As a result, sealing alone does not provide a great deal of confidence as a long-term solution to radon entry. However, sealing at least the major openings is almost a necessity for most other mitigation procedures, which are effective when pressure gradients are the primary driving forces. Without sealing, for instance, too great an airflow may be required to reverse the pressure gradient from the soil to the inside.

Ensuring that the airflow is in the direction from the house into the soil can be accomplished through a number of methods such as 1) sub-slab ventilation, 2) block wall ventilation, 3) baseboard ventilation, 4) drain tile ventilation, or 5) basement pressurization. Active sub-slab ventilation consists of using a fan to sweep the soil gas out of the aggregate under the slab in a basement or a slab-on-grade before it can enter the house through cracks or other pathways. In a typical implementation, one or more pipes penetrate through the slab into the aggregate and a fan withdraws air at a rate sufficient to reduce the local pressure to a value lower than that inside the house. Under these conditions, air flows from the house into the aggregate, thus preventing the entry of radon-bearing soil gas. If there are major unsealed openings in the floor, the energy penalty for heat losses will be prohibitive. This method will be less effective if the permeability of the aggregate or soil under the slab is low.

The centers of concrete blocks used to construct many basement walls contain voids, which are generally interconnected both vertically and horizontally within the wall, forming a network of channels through which air can move. Soil gas that penetrates the outer surface of the blocks through mortar joint cracks or pores is able to move throughout the wall to find cracks or openings to enter the basement. In the active ventilation of hollow-block basement walls, a fan is used to sweep the soil gas out of the voids in the wall and reduce the local pressure below that inside the basement so that the direction of airflow is from the basement into the wall. In this manner, radon is prevented from entering the basement. The typical implementation of this method is to install one or two pipes in each wall penetrating into a block cavity. However, there is another approach to implementing this method known as baseboard ventilation. In this case, a sheet metal "baseboard" is installed around the entire perimeter of the basement (including interior block walls), and covers the joint between the floor and wall. Holes are drilled through the interior face of the blocks at fixed intervals inside this baseboard, and the wall is ventilated by depressurizing the baseboard duct with a fan. This approach produces more uniform ventilation of the wall but is more expensive than the method that uses individual pipes. All major openings must be sealed for this method to be effective.

An alternative method for ventilation under the slab is called drain-tile soil ventilation. Perforated drain tiles surround part or all of some houses in the vicinity of the footing to drain moisture away from the foundation. The water is usually routed to an above-grade soakaway or to a sump in the basement. In many houses, the major radon entry points are near the floor-wall joint so that pumping on the drain tile near the foundation is often effective in removing the soil gas containing the radon. In many cases, the pressure near the foundation can be reduced sufficiently so that the airflow is reversed to move from the basement into the soil. When the aggregate under the slab is sufficiently permeable, the soil gas can be extracted from underneath the entire slab.

Still another method that ensures that the air flows from the basement into the soil uses basement pressurization. This pressurization can be accomplished by either blowing outside air into the basement or by blowing air from some other part of the house into the basement. This choice depends to some extent upon the outside temperature extremes and the amount of time the residents spend in the basement. This method would only be practical for houses in which the basement is well isolated from the remainder of the house and when combustion appliances are not adversely affected by the pressurization. Unless the basement is very tight, a severe energy penalty for heat loss is likely to occur.

The previously described techniques of ventilation were designed primarily for houses with basements or slabs-on-grade. In fact, it is thought that these types of construction will present the greatest number of problems because they represent cases in which the house is in intimate contact with the soil, providing an excellent opportunity for pockets of radon-bearing soil gas to collect and migrate into the house. However, in the case of crawl spaces that are not well ventilated, a similar situation could develop. Since the house tends to be depressurized (especially during winter) radon bearing soil gas in the crawl space will migrate into the house under the action of the pressure gradient. One approach to prevent radon entry is to reverse the pressure gradient through depressurization of the crawl space. An alternative approach is to pressurize the crawl space, thus preventing the radon from entering the crawl space and isolating the house from communication with the soil. In moderate climates, the most practical approach seems to be to adequately ventilate the crawl space (either actively or passively) by flushing or diluting the crawl space air with outside air.

Aside from switching to alternative water supplies or reconstructing the wells, the most effective methods for reducing radon levels in water supplies seem to involve aeration or adsorption by granular activated carbon. Activated carbon is quite effective in reducing the radon level, but has two potentially significant drawbacks. First, the granular beds show a tendency to develop bacterial growths which may eventually cause health concerns. Second, the granular bed adsorbs radioactive components (uranium, radium, radon, and RDPs from the water and, consequently, becomes radioactive. One result is increased gamma radiation in the vicinity. It presently is not clear whether this increased gamma radiation creates a significant danger, but the matter should be studied. Another result is that the accumulation of uranium and radium in the bed may cause the material to be classified as a low-level radioactive waste and, consequently, create a serious disposal problem. Aeration is another effective means of removing radon from water. The most common method involves breaking the water into small droplets as a spray. The radon readily diffuses from the liquid into air.

2.8 BIOLOGICAL CONTAMINANTS

2.8.1 Introduction

Indoor environments are contaminated by a variety of biological pollutants. Among these are molds and their spores and toxins, bacteria, viruses, protozoans, algae, body parts and excreta of insects, acarids, and arachnids dander and excreta from animals, and pollens from higher plants.

Airborne molds most commonly found in indoor environments include the following:

Aspergillus
Penicillium
Fusarium
Cladosporium
Aureobasidium
Sporobolomyces
Wallemia
Yeasts
Thermophilic *Actinomycetes*

Aspergillus, *Penicillium*, *Fusarium*, and *Cladosporium* are known toxigenic taxa (Burge, 1986). Among the bacteria that have been identified indoors are *Legionella pneumophila*, *Clostridium perfringens*, *Staphyllococcus aureus* and *S. dermatitis*, *Streptococcus pyogenes*, and *Salmonella typhimurium*. *Mycobacterium* species have been found in the dust of homes of infected persons. Occasionally *Pseudomonas* species and *Actinobacter* species have been found. A number of other bacteria associated specifically with factories processing organic materials from plants or animals have been isolated in those environments (Solomon and Burge, 1984; Imperato, 1981; Sugarawa and Yoshizawa, 1984; Lundholm and Laurell, 1984). Among those viruses transmitted by the airborne route are smallpox (no longer considered to be present as an infectious agent outside of tightly controlled laboratories), chickenpox, measles, rubella, influenza, adenovirus 4 and 7, coxsackie A21, and lymptocytic choriomeningitis viruses (Couch, 1981).

Protozoa, especially amoeba of the genera Acanthamoeba, Naegleria gruberi, and N. fowleri, have been identified indoors, on occasion. Algae, especially algal spores, have been identified in dust.

Microscopic body parts and excreta of insects and acarids tend to be found in house dust. Dust mites, Dermatophagoides spp., themselves nearly of microscopic size, produce fecal pellets, which disintegrate to form particles in the respirable size range (0.8 to 1.4 μ), which can be highly allergenic in sensitized individuals (Andersen and Korsgaard, 1986). Dried excreta of insects such as cockroaches can also be entrained in dust and be a source of allergy. Larger animals such as dogs, cats, birds kept as pets, as well as rodents, can produce a number of body secretions as well as skin flakes, that serve as allergenic agents.

Pollens from higher plants enter indoor spaces from outside. Pollens from indoor plants are not generally considered a problem.

2.8.2 Sources of Biological Contaminants

Sources of biological contaminants vary greatly. Many have their origin out of doors and enter indoor spaces through windows, doors, and cracks, or through ventilation, and air conditioning or humidifying systems. Animal dander and excreta, especially from birds such as pigeons roosting near air inlets of buildings, can also enter the indoors. Among contaminants originating outside are pollens. These generally are windborne pollens from small, rather drab, fragranceless plants, unlike houseplants grown for showy or fragrant flowers. Their concentrations vary seasonally, and indoors additionally with indoor/outdoor pressure differentials, amount and type of ventilation, wind conditions, temperature, and humidity (Burge, 1985). Algae and amoeba also enter from outside as do fungi, in similar fashion. These latter are viable, however, and can colonize suitable indoor environments, increasing tremendously in number once established in a friendly space indoors. House dust mites, which feed on skin flakes shed by humans and animals, can also multiply under ideal conditions and hence increase their output of allergenic products. Ideal conditions for increased growth among these organisms are moisture, humid air, and warm temperatures (Burge, 1985). Any organic material can serve as a medium for fungal growth. Moist surfaces and water reservoirs for air conditioning and humidifying systems for homes and buildings can serve as ideal reservoirs, amphifers and disseminators for fungi, bacteria, algae and amoeba. Office and public buildings that use chilled water air conditioners/humidifiers have been identified as one building type that is subject to such contamination. *Legionella* ssp. and *Clostridium* ssp. are soil bacteria that have been found in the reservoirs of such systems. In addition to such a water source for bacteria, humans expel saprophytic and pathogenic bacteria by sneezing, coughing, and speaking, and can introduce such organisms into indoor spaces. These bacteria may survive for significant periods of time depending on droplet size, temperature, and relative humidity. Such bacteria, as well as fungal spores and amoeba droplet nuclei, and sometimes viruses, have been distributed throughout buildings by ventilating systems, stairway and elevator shafts through stack effects, and even through building raceways and plenums. In homes, many of these biological contaminants can likewise be found, and home air conditioners have sometimes been identified as the source of the organisms. Growth indoors is promoted by moisture and organic food sources, so that upholstery, bedding, carpets, plumbing, and food preparation areas tend to be sources of continuing contamination.

The two most common fungi appearing indoors are *Penicillium* and *Aspergillus* (Benson et al., 1972). Although measured concentrations of fungus spores are generally less indoors than outdoors, under poor hygienic conditions and/or conditions of high humidity, fungal spore concentrations indoors have been measured which exceed outdoor concentrations by 200 to 400 percent (Benson et al., 1972; Nilsby, 1949; Flensborg and Samsoe-Jensen, 1950; Jimenez-Diaz et al., 1960; Solomon, 1975). Outside sources of fungi, as well as of bacteria, are humidifiers and cooling towers associated with air conditioning systems (Miller et al., 1976; Taylor et al., 1978; National Research Council, 1981b).

Indoor concentrations of pollen, known to cause allergic reactions in sensitive individuals, are driven by outdoor concentrations. The pollen enters through building cracks and crevices, doors, windows, and the fresh-air intake of air conditioning systems.

Epidemiological studies have established that many infectious diseases are communicated by airborne transmission (Repace, 1982). The well-known Legionnaire's disease is a good example of how outside contamination can affect the occupants in a building. The disease-causing organism, *Legionella pneumophilia*, is unusual among pathogens in that it apparently exists in outdoor natural reservoirs (soils), and infection is possible through inhalation of contaminated outdoor air (National Research Council, 1981b).

2.8.3 Monitoring of Biological Contaminants

Sampling and measurement of biologic pollutants present special complications not found with less complex chemical agents. Devising universally suitable means of determining the presence and concentration of biological contaminants remains a problem.

In the United States, the most common method for sampling biological particles remains gravity collection, either on culture plates or sticky slides. This method is never volumetric, produces a qualitatively biased picture of the air spora, and should never be used for assessment of airborne particle levels. It is useful only where gravity deposition of particles is of concern (such as in operating rooms).

Volumetric sampling (where a measured quantity of air is collected and analyzed) includes two general types: 1) viable or cultural methods where recovery depends on the viability of collected particles as well as their ability to grow under given conditions, and 2) particulate methods where particles are visually counted and identified or biochemically or immunologically analyzed.

Cultural methods are useful when 1) information on viability is essential (e.g., for infectious agents); and 2) particles must be cultured to be identified (e.g., actinomycetes, bacteria, viruses, many small-spored fungi). Choice of culture media for these samplers is critical. Cultural methods always underestimate actual spore counts, and the underestimate increases logrithmically with particle levels (Burge et al., 1977).

Particulate sampling is the method of choice when total biological particle counts are to be assessed or when biological products (toxins, antigens) are to be measured. Total fungal spore counts can be simply done using microscopic counting. New methods using fluorescent staining may enable counting of bacterial particles as well (Palmgran et al., 1986). Immunological or biochemical analysis of particulate samples is especially useful when known contaminants are to be analyzed. These methods have been used for airborne endotoxin (Rylander and Haglind, 1984), mycotoxin (Burg et al., 1981), and a variety of antigens (e.g., mites (Swanson et al., 1985), cockroaches (Swanson et al., 1985), and thermophilic actinomycetes (Reed et al., 1983)). Sampling for particles to be analyzed biochemically or immunologically requires advance ledge of the compounds of interest. These methods are not useful for surveys of general bioaerosol contamination.

Volumetric collection modalities fall into three general categories: impactors, impingers, and sieve samplers (Burge and Solomon, 1987). Impactors collect by causing particles to leave the air stream and impact on an adhesive surface either by rapidly rotating the sampling surface or by accelerating the air. Commonly used impaction samplers are listed in Table 2-41. The rotating and centrifugal impactors are efficient only for particles larger than 15 to 20 µm. Remaining samplers are efficient over a wide range of particle diameters.

TABLE 2-41. IMPACTION SAMPLERS USEFUL FOR BIOAEROSOL SAMPLING

Sample Type	Viable	Particulate
Rotating impactors		Rotorod[a], Rotoslide
"Centrifugal" samplers	RCS[b], Wells	
Single plate impactors	Andersen-N6[c], SAS[d], Microban[e]	
Cascade impactors	Andersen	Andersen
Slit samplers	Slit sampler[f]	Burkard[g]

[a] Ted Brown Assoc., Palo Alto, CA

[b] Biotest Diagnostics, Fairfield, NJ

[c] Andersen Samplers Inc., Atlanta, GA

[d] Spiral Air Systems, Bethesda, MD

[e] Ross Industries, Midland, VA

[f] New Brunswick Scientific Co., New Brunswick, NJ

[g] Burkard Manufacturing Co., Rickmansworth, England

Impingers trap particles in a liquid after impingement on a submerged surface. The resulting collection fluid can be analyzed immunologically or biochemically. Liquid impingers are especially useful for sampling for viable particles in highly concentrated aerosol situations because the fluid can be dilution cultured. The use of these devices for fungi, toxins, and antigens has not been well studied.

Sieve sampling utilizes filters of known pore sizes either in portable filter cassettes or in larger high-volume sampling devices. Filters collect particles to diameters well below rated pore sizes very efficiently. Samples can be analyzed by direct counts on the filter, or by cultural, biochemical, or immunological assay of filter eluates.

At present, no one sampler is adequate for all bioaerosols. A combination of a cultural sampler and a particulate collector will cover most situations. Both filtration and impinger sampling have the potential of being universal samplers, but much research remains to be done to verify their use for all bioaerosol types.

2.8.4 Health Effects of Biological Contaminants

2.8.4.1 Infection. Health effects of biological contaminants can be described primarily as three kinds: pathogenic, toxicogenic, and allergenic. Some organisms are clearly infective, and their entry into human respiratory systems presents the possibility of disease. The syndrome for each disease entity is usually well defined and often leads to identification of the infective agent. Viral diseases known to be spread by the airborne route, such as measles, rubella, chickenpox (varicella), are clearly identified. Respiratory diseases such as colds, influenza, and sore throats may be less clearly defined since some symptoms are similar to allergic responses. Viruses are different from bacteria and other infective agents because they cannot replicate outside of their host and they are highly species-specific.

A fairly common soil bacterium, which grows well in organically enriched water, and which is disseminated in airborne droplet nuclei, is *Legionella* spp. It is the cause of a serious and sometimes fatal pneumonia known as Legionaires disease. Epidemiologic studies have shown that *Legionella* causes 10 to 15 percent of pneumonia cases in communities and in hospital situations (Johnson-Lussenburg, 1986). More commonly it causes a milder, flu-like syndrome termed Pontiac fever, that disappears without medical treatment.

Other waterborne bacteria such as *Acinobacter* and *Pseudomonas aeruginosa* can become airborne and be disseminated through heating, ventilation, and air conditioning (HVAC) systems. This can be a hazard in hospitals, where severely immuno-compromised patients may develop pneumonia from airway colonization by these bacteria, which are not pathogenic to normal immunologically competent persons (Tobin, 1986). Burn patients may also develop severe wound infections from these organisms.

Mycobacterium tuberculosis is an extremely virulent bacterial organism which can cause infection if only a single droplet nucleus is inhaled (Riley, 1982). Its source is infected humans. *Mycobacterium* is encapsulated and may survive for fairly long time periods in dried sputum, and may become reentrained in air streams in buildings.

Some saprophytic fungi can behave opportunistically and become invasive human pathogens. Systematic studies of such occurrence do not exist, but some data are available from general surveys of indoor microfungi. *Aspergillus fumigatus*, *Mucor*, and *Absidia* species are common in indoor areas involving hay handling (Solomon and Burge, 1984), while *Phialophora* and *Fusarium*, which have taxa that include human pathogens, have been isolated from humidifier fluid. Yeasts such as *Sporothrix* and *Geotrichum* are found colonizing cool-mist vaporizers used indoors, and contaminate the air copiously during operation (Solomon and Burge, 1984).

Human pathogens such as *Blastomyces*, *Crypotococcus*, *Coccidioides*, and *Histoplasma* are saprophytic in natural reservoirs of bird and animal droppings and enter the body by the respiratory route, causing respiratory and systemic infections. Systemic mycoses begin as lung infections, but the fungus can migrate to other organs including the heart, brain, and kidneys. For instance, disseminated blastomycosis may include liver, spleen, and long bones; coccidiomycoses cases can result in meningitis, spondylitis, and otomycoses; histoplasmosis can range from flu-like symptoms to ocular histoplasmosis and acute disseminated histoplasmosis (Day, 1986). Such serious invasive disease is rare. However, acute disseminated histoplasmosis is usually fatal in children. Systemic mycoses have been reported in individuals exposed to air from contaminated ventilation systems. *Coccidiodes* and *Histoplasma* are considered highly infective. Indoor contamination from these fungi is not known (Solomon and Burge, 1984). Soil contaminated with animal droppings containing these organisms is readily reentrained, especially during windy conditions in arid regions, and epidemics of *Coccidioides* infection have occurred during dust storms (Ajello et al., 1965; Flynn et al., 1979). *Blastomyces* and *Histoplasma* likewise can be dispersed through reentrained contaminated soils, and their spores can penetrate indoor spaces (Ajello, 1967).

2.8.4.2 *Mycointoxication*. Species-specific information regarding fungi is relatively sparse, but essential if health effects of the organism are to be described. Some fungi are pathogenic in humans, and their presence alone is cause for concern. Some fungi also produce potent mycotoxins. Effects of these poisons are primarily known from their ingestion, and information concerning toxic potential via inhalation is practically unknown. The best known example is an isolate of *Aspergillus flavus* which produces aflatoxin. Aflatoxins are among the most potent liver toxins and carcinogens presently known

(Rodricks et al., 1977). Three different aflatoxins (B_1, B_2, and G_1), are known animal carcinogens (Nesnow et al., 1986). Aflatoxin B_1 is the most studied of the three. The mutagenic action of Aflatoxin B_1 has been studied and found genotoxic in many microorganisms, plant, and animal test systems (Ong, 1975). For example, Aflatoxin B_1 is mutagenic to Salmonella typhimurium (Stark et al., 1979), produces micronuclei in bone marrow erythroblasts of rats and mice (Trzos et al., 1978; Friedman and Staub, 1977), and increases sister chromatid exchanges (SCEs) in mouse bone marrow cells (Nakanishi and Schneider, 1979) and chromosome aberrations in cultured human lymphocytes and Chinese hamster cells (Dolimpio et al., 1968).

The spores of toxigenic fungi contain mycotoxins, often at very high concentrations. It is reasonable to assume that these toxins would have a systemic effect when inhaled, since the inhalation route more effectively allows systemic entry for dissolved substances than ingestion does. Perhaps 30 to 70 percent of isolates tested appropriately are known to produce toxins, and isolation of a given species does not necessarily mean that isolate is toxigenic. While that can only be determined by testing, it is prudent to assume a toxigenic potential until proven otherwise (Tobin et al., 1986). Other Aspergillus species, such as A. niger, are not considered a threat to human health. Yet A. fumigatus and A. parasiticus are pathogenic, toxicogenic, and allergenic. Toxigenic fungi do not always produce their toxins, especially in vitro, but it is wise to assume that toxigenic strains produce their toxins in nature.

Molds such as Stachybotrys, Fusarium, Trichothecium, Trichderma, Aeromonium, Cylindocarpon, and Myrothenium are known to produce trichocene mycotoxins. Such toxins produce direct toxic effects as well as immunosuppression. At low concentrations they produce gastrointestinal lesions, hematopoietic suppression, and suppression of reproductive function. Rapidly growing cells seem to be affected most. Toxicity of the central nervous system produces symptoms such as anorexia, lassitude, and nausea. Vague symptoms such as those described in "building related illness" or SBS can be attributed to trichocene intoxication. Immune suppression can facilitate opportunistic infections by other molds or bacteria (Day, 1986).

An example of possible mycotoxin intoxication is that associated with the fungus Stachybotrys. This is a saprophytic fungus that grows on high-cellulose media low in nitrogen sources. It can grow on nutrient-poor substrates such as dust, shower curtains, straw, and wallpaper. It requires moisture and lack of competition from vigorous fungi (Jarvis, 1986).

Jarvis (1986) describes a case study of an airborne outbreak of Stachybotryotoxicosis in Chicago, IL. A family occupying a brick home in which a heavily contaminated cold air return duct and a wood fiber board (Celotex^{TM}, Chicago, IL) ceiling insulation material demonstrated heavy growth of *Stachybotrys atra*, complained of recurring symptoms. Among these were cold and flu symptoms, sore throats, diarrhea, headaches, fatigue, dermatitis, intermittent focal alopecia, and generalized malaise. The father experienced severe leg pains. Some family members showed signs of psychopathy, and one of the teenaged sons committed suicide. Repeated hospital and clinical examination revealed no causes of these symptoms.

Air in the home was contaminated with spores from *Stachybotrys atra*, and tests for trichocene mycotoxin were positive. Trichocenes were also isolated from samples of the fungus contaminating the duct and the ceiling insulation board. Included were the toxins verrucarin B and trichoverrines A and B. After the Chicago house was thoroughly cleaned of the *S. atra* - contaminated ducts, insulation and ceiling material, the family reoccupied the house and no longer suffered from the earlier complaints. It is possible to correlate their symptoms with trychocene intoxication, specifically those isolated from the house air, and known to be produced by *S. atra*.

2.8.4.3 *Allergenic Reactions*. More commonly, fungi are associated with allergenic reactions indoors, which can vary in intensity and type, and can be described as follows:

2.8.4.3.1 *Allergic rhinitis*. This is often termed "hay fever" when seasonally related, which involves nasal air passage obstruction and itching, sneezing, and oversecretion of mucus. Conjunctivitis, which involves irritation, itching, and reddening of the eyes, is often associated. Excessive mucus secretion and blocking of sinus and eustachian passages provide growth reservoirs where secondary bacterial infections may implant.

2.8.4.3.2 *Bronchial asthma*. This disease, which involves a recurrent narrowing of bronchioles and hypersecretion of thick mucus that can block airways, is accompanied by varying degrees of wheezing, shortness of breath, and coughing. Secondary bacterial infections can result in bronchitis and more sensitive reactions to irritants and other allergens.

2.8.4.3.3 Hypersensitivity pneumonitis (extrinsic allergic alveolitis). This ailment is the most serious acute immune reaction to sensitizing substances. It involves the production of large amounts of IgG antibody, cellular hypersensitivity, and the formation of interstitial granulomas. It causes filling and variable destruction of the alveoli by inflammatory cells. With continued exposure irreversible pulmonary fibrosis and eventual pulmonary failure, ending in death, ensues (Reed, 1981; Solomon and Burge, 1981).

2.8.4.3.4 "Monday complaints." This describes a syndrome generally occurring on return to a work environment after one or more days' absence. It may be related to hypersensitivity pneumonitis. Symptoms and signs include air hunger, wheezing, coughing, fever, and muscle pains. These flu-like symptoms appear after hours in the sensitizing environment, often last overnight and recur, with decreasing severity, on sequential daily exposure. Long-term involvement is associated with chronic bronchitis and with lung scarring (Solomon and Burge, 1981).

2.8.4.4 Other Allergens. While the sensitizing agents for these described illnesses frequently are molds, other biological entities can provoke any of these symptoms. Nonviable agents such as house dust, mite fecal pellets, cockroach feces, insect and arachnid dried hulks and body parts, animal danders, nonviable remains of molds and their spores, dried, reentrained animal excretions such as saliva, sweat, urine and feces, pollens, and biogenic volatiles, have also been identified as actors. Among the viable organisms provoking such responses are molds, amoebae, algae, and actinomycetes.

Amoebic species which have been implicated in hypersensitivity pneumonitis are *Acanthamoeba* spp. and *Naegleria gruberi*, and these have also been isolated from humidifier baffle plates and from humidifier waters in homes and factories (Sykora et al., 1982). *Acanthamoeba* infection of the cornea among contact lens wearers with resulting sclerokeratitis and epithelial erosion has recently been reported (Mannis et al., 1986), and may be related to air contamination from air conditioning/humidifying systems.

2.8.5 Indoor Air Quality Control Options

Many indoor biological contaminants may be greatly reduced by humidity control and cleanliness. These practices include:

1. Humidity control in homes and buildings at a level not conductive to the growth of fungi, molds, and dust mites. This is generally in the 45 to 50 percent relative humidity (RH) range. As indicated previously, dust mites will not survive below 45 percent RH.
2. Periodic and thorough cleaning of all places where water is likely to collect. These places include: drip pans of humidifiers and refrigerators, cooling towers, and in and around toilets and wash basins.
3. Periodic and thorough cleaning of carpets and fabrics.
4. Extermination of household insect pests.
5. Periodic cleaning or replacement of furnace and air conditioning filters and other air cleaning devices.

2.9 PESTICIDES

2.9.1 Introduction

Pesticides (including, among others, insecticides, rodenticides, termiticides, and germicides) are used both by professional pest control businesses and by occupants of indoor spaces to eliminate a wide variety of organisms, ranging from rodents to insects, fungi, bacteria, and viruses. They are by definition poisons, but their range of toxicity varies with their target. They include dicoumarins, organophosphates, carbamates, and chlorinated hydrocarbons, among others. Their use, storage, and disposal are, for the most part, regulated, but cautionary information to homeowners, in particular, must be clarified and emphasized.

2.9.2 Sources of Pesticide Exposure

A national household pesticide usage study involving over 8,000 households was conducted by EPA (U.S. Environmental Protection Agency, 1980) in 1976 and 1977. It is estimated that 94 percent of pesticide usage is for agricultural purposes. Pesticides, herbicides, and fumigants used in agriculture may become airborne and attach themselves to airborne particulate matter, or, through soil runoff, contaminate rivers, lakes, and streams which may be a source of water supplies. A study on the role of house dust in, for example, DDT pollution (Davies, 1972), indicates that house dust can be a principal source of insecticides. The concentrations of DDT in house dust was higher than that found in soils in the area. Table 2-42 shows that there are many sources

TABLE 2-42. SOURCE OF INDOOR PESTICIDE EXPOSURE

Compound (Trade Name)	Type of Pesticide	Household Uses Leading to Potential Human Exposure
Chlorpyrifos (Dursban®)	Insecticide	Control of mosquitoes, cockroaches and other household insects; turf and ornamental insects; fire ants, termites, and lice
Pentachlorophenol	Fungicide Insecticide	Exterior wood preservative
Chlordane	Insecticide	Subterranean termite control
Ortho-Phenylphenol	Disinfectant, Fungicide	Household disinfectant; post-harvest application to fruits and vegetables
Propoxur (Baygon®)	Insecticide	Control of cockroaches, flies, mosquitoes; lawn and turf insects
Resmethrin	Insecticide	Control of flying and and crawling insects; fabric protection; pet sprays and shampoos; application on horses and in horse stables; greenhouse use
Dicofol	Insecticide	Control of mites on fruit, vegetable, and ornamental crops
Captan	Fungicide	Seed protectant; fungal control on fruits, vegetables, and berries
Carbaryl (Sevin®)	Insecticide	Control of insects on lawns, ornamentals, shade trees, vegetables, and pets
Lindane (γ-BHC)	Insecticide	Seed treatment; insect control in soil, on vegetables, ornamentals and fruit and nut trees
Dichlorvos (DDVP)	Insecticide	Household and public health insect control; flea collars and no-pest strips

(continued on following page)

TABLE 2-42. (continued)

Compound (Trade Name)	Type of Pesticide	Household Uses Leading to Potential Human Exposure
2,4-D esters	Herbicide	Post-emergent weed control
Malathion	Insecticide	Insect control on fruits, vegetables, ornamentals, and inside homes
Permethrin (cis and trans)	Insecticide	Control of flies, mosquitoes, ants, cockroaches, garden insects
Heptachlor	Insecticide	Subterranean termite control
Aldrin	Insecticide	Subterranean termite control
Dieldrin	Insecticide	Subterranean termite control
Ronnel	Insecticide	Fly and cockroach control
Diazinon	Insecticide, Nematicide	Control of soil and household insects, grubs and nematodes in turf; seed treatment and fly control
Methoxychlor	Insecticide	Control of insects in garden, fruit, and shade trees
Atrazine	Herbicide	Weed control
α-Hexachlorocyclohexane (α-BHC)	Insecticide	Manufacture and use discontinued in U.S.; ubiquitous in air, residue from lindane
Bendiocarb (Ficam®)	Insecticide	Household, ornamental, and turf insect control
Folpet	Fungicide	Fungus control on flowers, ornamentals, seeds, plant beds; paints and plastics
Chlorothalonil (Bravo©)	Fungicide	Broad spectrum fungicide; wood preservative; paint additive
Dacthal	Herbicide	Selective pre-emergent weed control on turf, ornamentals, and vegetable crops

(continued on following page)

TABLE 2-42. (continued)

Compound	Type of Pesticide	Household Uses Leading to Potential Human Exposure
Oxychlordane	--	Oxidation product of chlordane
Heptachlor epoxide	--	Oxidation product of heptachlor
trans-Nonachlor	--	Component of chlordane
PCBs (Aroclors 1242 and 1260)	--	Used in electrical transformers until 1976

Source: Lewis et al. (1986).

leading to indoor exposures of pesticides (Lewis et al., 1986). As indicated in Table 2-42, many pesticides are used directly in the indoor environment or in close proximity (e.g., foundation treatment for termites). Chlordane has been detected in the air of some termiticide-treated homes as long as 14 years after application (Repace, 1982; Staats, 1980). It is also well recognized that many insecticides and herbicides find their way into the home through the food chain.

2.9.2.1 Emission Rates. While many studies have been conducted to determine the indoor concentrations of pesticides, limited data are available on emission rates of pesticides to the indoor environment. Leidy et al. (1984) showed that emissions decreased with time for diazinon applied in cracks and crevices in a dormitory. Jackson and Lewis (1981) evaluated emissions of propoxur, diazinon, and chlorpyrifos from pest control strips. Nelms et al. (1987) conducted tests in environmental test chambers of paradichlorobenzene emissions from moth crystal cakes at two temperatures and four air exchange rates. Estimates of the emissions rates are shown in the second and third columns of Table 2-43.

2.9.3 Exposure to Pesticides

To further define the levels of pesticides occurring in indoor air and the potential health hazard resulting from such exposure, the U.S. Environmental Protection Agency's Office of Pesticide Programs is developing and implementing guidelines that will require that pesticide manufacturers submit indoor respiratory exposure and dermal exposure information for those pesticide products

TABLE 2-43. EMISSION RATES OF PARADICHLOROBENZENE FROM MOTH CRYSTAL CAKES (mg/cm² hr)

Air Exchange Rate (hr-1)	Temp. = 23°C	Temp. = 35°C
0.25	1.2	4.5
0.5	1.3	4.7
1.0	1.7	5.6
2.0	2.0	6.3

Note that temperature had a marked effect on the emission rates.
Also, the chamber air exchange rate impacted the emission rates due to the suppression of evaporation at low air exchange rates due to high chamber concentrations.

used indoors. Currently, indoor air monitoring data are required only for those pesticides (e.g., aldrin, dieldrin, chlordane, heptachlor, chlorpyrifos) registered for termite control. According to the draft Pesticide Assessment Guidelines, Subdivision U, Applicator Exposure Monitoring of January 2, 1986, inhalation exposure generally represents a very small component of the total exposure; however, the application of dusts, aerosols, and fumigants, or the application of sprays in enclosed spaces can result in significant inhalation exposure. Therefore, respiratory exposure information will be required for those pesticide products used indoors for which (a) the toxicological evaluation indicates that the use of the product may pose an acute or chronic hazard to human health, (b) inhalation exposure is likely to occur during use and/or for a period of time after use, (c) data are not available for estimating the magnitude of exposure for a particular work activity and postwork activity with an acceptable degree of confidence, and (d) depending on the physiochemical properties of the product, the conditions of use, and the toxicological effects, it can be expected that exposure by inhalation will be of significant concern as compared to exposure by dermal absorption. Indoor sites include, but are not limited to: homes and apartments, greenhouses, barns and other farm buildings, commercial buildings and manufacturing facilities, restaurants and food handling and processing facilities; fumigation facilities, warehouses, railroad boxcars, schools, hospitals and other health care facilities, and mushroom houses.

2.9.4 Monitoring

Interest in measuring pesticide exposure in indoor air is relatively recent. Few studies on pesticides were conducted in the 1970s, with perhaps the earliest published work being that of Starr et al. (1974), which describes measurements in Colorado and concludes that household dust is a major reservoir for pesticides in the indoor environment. Jackson and Wright (1975) evaluated the variance in pesticide residues following application using air compressors and aerosol-type sprayers. They concluded that less pesticide movement occurred following aerosol application. They also determined that food contamination after insecticide application was appreciable.

Wright et al. (1981, 1984); Leidy et al. (1982, 1984), and Wright and Leidy (1982) published a series of papers describing measurements following application of pesticides in dormitories, food-serving areas, buildings, service vehicles, and homes. In each case, measurable amounts were detected in the air, on dust particles, and on the walls, and articles in the rooms, for as long as 35 days after application.

Pellizzari et al. (1981), Jurinski (1984), Ruh et al. (1984), Gebefuegi and Korte (1984), Livingston and Jones (1981), and Dobbs and Williams (1983) also reported measurable pesticide exposure after residential or commercial application of pesticides. Melius et al. (1984) published a survey by the National Institute for Occupational Safety and Health (NIOSH) of more than 200 buildings in which office workers had complained of poor indoor air quality. Pesticides may be a major contributor to the sick building syndrome in some of these cases.

Increased attention is being paid to improving the methodology for measuring pesticides in indoor air, largely because agencies such as EPA are required to measure and monitor low levels of pesticides in the indoor environment. Melcher et al. (1978), Lewis et al. (1986), and Jackson and Lewis (1981) identified methods for increasing sampling sensitivity for a variety of chemicals. It is now possible to measure some air pesticide concentrations as low as 0.01 $\mu g/m^3$.

A battery-powered, low-volume (4 L/min) sampler suitable for indoor air and personal exposure monitoring was developed in the early 1980s (Lewis and MacLeod, 1982). The sorbent found most convenient to use in this sampler was polyurethane foam (PUF). Sampling efficiencies have been determined for 58 organochlorine, organophosphate, organonitrogen and pyrethroid pesticides, as well as for several polychlorinated biphenyl (PCB) mixtures. The sampler was successfully field-evaluated in 1985 in a pilot test by the Agency's Non-occupational Pesticides Exposure Study (NOPES) methodology (Lewis et al., 1986; Lewis et al., 1987). The sampler is currently being used in the two-city, 300-home NOPES project. Analytical problems were encountered, however, with two common household pesticides, acephate and glysophate, so these compounds could not be included in the NOPES program.

PUF may also be used to collect other SVOCs, but breakthrough problems occur with SVOCs having vapor pressures greater than 10^{-3}mm Hg. A combination sorbent trap in which Tenax GC or other granular sorbent is sandwiched between two PUF plugs has been demonstrated to efficiently trap the more volatile SVOCs (Lewis and MacLeod, 1982). However, this system has not been fully validated in the laboratory or in the field.

The major requirement in a methodology for monitoring low levels of pesticides exposure is the ability to sample with the precision and accuracy needed for regulatory purposes. While technology is available to absorb, elute, and measure some of the pesticides now in use, no field-tested, quantitatively valid methods are available for testing many others. Methods with known precision and accuracy are needed to establish exposure measurement guidelines based on field monitoring (i.e., sampling and analysis).

Out of concern for the lack of information on nonoccupational exposure to pesticides, Congress in 1985 passed a bill that provided funds for a total pesticide exposure assessment study to be conducted by the U.S. Environmental Protection Agency.

In response to this initiative, ORD developed the NOPES project. To date, a pilot study of nine single-family dwellings has been completed, and Phase I of the NOPES project is under way. The purpose of the pilot study was to select, validate, and field test sampling methodologies and survey questionnaires to be used in preparation for the NOPES project. Most of the households selected for the pilot study belonged to retired or semiretired persons who generally spend an average of 18 hours indoors daily. Thirty pesticides and related chemicals were selected for monitoring based on present day and historical usage (Table 2-42). Indoor, outdoor, and personal exposure air monitoring and tap water analyses were performed at all of the dwellings. Of the 30 pesticides and related chemicals tested, 22 were detected in indoor and/or outdoor air samples (Table 2-44). The concentration of pesticides occurring indoors was generally higher than the levels found outdoors, with peak values ranging from 1.7 to 15.0 $\mu g/m^3$, compared to 0.001 to 0.41 $\mu g/m^3$, respectively. Table 2-45 lists the most commonly found pesticides and their

TABLE 2-44. SUMMARY OF PESTICIDES FOUND IN AIR FROM NOPES

Pesticide	Number of Households at Which Pesticides Were Detected in Air: Indoors	Outdoors	Respiratory
Chlorpyrifos	9	7	8
Diazinon	8	7	6
Chlordane	8	6	6
Propoxur	7	4	6
Heptachlor	7	5	6
trans-Nonachlor	5	3	5
o-Phenylphenol	5	4	5
Dieldrin	5	3	5
Lindane	5	2	3
Aldrin	4	4	3
Ronnel	4	1	3
Captan	4	1	3
Bendiocarb	3	1	3
α-Hexachlorocyclohexane	3	2	3
Folpet	3	4	3
Malathion	3	2	1
Chlorothalonil	3	3	1
Dichlorvos	2	1	0
Dicofol	1	2	0
Methoxychlor	1	1	0
Pentachlorophenol	1	1	0
trans-Permethrin	0	1	0

Source: Lewis et al. (1986).

TABLE 2-45. SUMMARY OF MONITORING DATA FOR THE FIVE MOST PREVALENT PESTICIDES (NOPES)

Pesticide	Indoor ($\mu g/m^3$)	Outdoor ($\mu g/m^3$)	Personal Exposure ($\mu g/m^3$)
Chlorpyrifos	0.014 to 15	ND to 0.30	ND to 8.8
Diazinon	ND to 8.8	ND to 0.41	ND to 5.1
Chlordane	ND to 1.7	ND to 0.21	ND to 4.2
Propoxur	ND to 0.66	ND to 0.0039	ND to 0.6
Heptachlor	ND to 0.31	ND to to 0.048	ND to 0.18

*ND = Not detected at ca. 0.001 $\mu g/m^3$.

Source: Lewis et al. (1986).

monitored exposure levels. In general, these pesticides were ingredients of pesticide products found in the house or recently applied by the occupants or professional pest control operators (Lewis et al., 1986).

Phase I of the NOPES project involves indoor and outdoor air monitoring of 70 dwellings in Jacksonville, FL. The air monitoring is scheduled to last for three seasons. Phase II of the NOPES project was to be initiated in December 1986. This portion of the study will encompass indoor and outdoor air monitoring of 50 dwellings in a northern city for two seasons.

2.9.5 Health Effects Associated with Pesticide Exposure

There is a battery of information available on the health effects of pesticides, ranging from acute to chronic exposure effects and carcinogenicity. However, this information is generally based on animal studies and covers those effects resulting from the oral or dermal routes of exposure. The limited information on inhalation exposure is mainly in the form of acute toxicity studies with actual exposure to the pesticide lasting approximately four hours. Information on human pesticide exposure via inhalation indoors is lacking, as are data from long-term animal inhalation studies.

2.9.6 Mitigation and Control Options

2.9.6.1 Ventilation. As with other indoor air pollutants, increased ventilation is effective in reducing pesticide concentrations via dilution and flushing, although limit data are available. Levin and Hahn (1986) showed increased ventilation to be effective in reducing levels of pentachlorophenol in a building with exposed inside wood beams that had been treated with this compound.

2.9.6.2 Air Cleaners. No references were found on the application of air cleaning devices for the control of indoor pesticide levels. Depending on the characteristics of the pesticide vapor, adsorption and/or catalytic oxidation may be applicable. Specific studies would be required.

2.9.6.3 Material/Product Selection and Use. Levels of pesticide residuals in the indoor environment can be controlled either by avoiding use or by using smaller amounts and less frequent applications. When a pesticide must be used, careful adherence to product label directions is essential. Many pesticides are controlled via registration and are available only through professional applicators. The methods of applying pesticides can also affect exposure.

Wright and Jackson (1975) indicate that aerosol-type sprayers are more effective than compressed-air sprayers in limiting the application to the target areas.

2.9.6.4 Other Measures. Indoor exposures to pesticides from treated wood can be reduced by sealing. Levin and Hahn (1986) report that pentachlorophenol levels are lowered when exposed beams are sealed with a polyurethane varnish, although the long-term effectiveness of this approach has not been determined.

2.10 NONIONIZING RADIATION: EXTREMELY LOW FREQUENCY ELECTRIC AND MAGNETIC FIELDS

2.10.1 Occurrence and Sources of Nonionizing Radiation

Electric power generation, transmission and distribution, and final utilization in homes, offices and factories is one hallmark of our modern industrialized society. In the United States, most electric power is in the form of electric current alternating at a frequency of 60 cycles per second (60 Hz), which falls into the extremely low frequency or ELF portion of the electromagnetic spectrum. Because of the high voltages and currents that occur at various stages from generation to endpoint utilization, there are attendant electric and magnetic fields that permeate the surrounding environment. Thus there are magnetic and electric fields generated in or near every electrified house, office building, and factory in this country, that expose the occupants to electromagnetic fields at the power line frequency. The same situation exists throughout the world, except the alternating current may be at a different frequency (most likely 50 Hz).

There are many sources of electric and magnetic fields commonly found indoors. These include any appliance that (1) has an electric motor (refrigerator, freezer, clothes washer, hair dryer, shaver, food mixer and chopper, clock, vacuum cleaner), (2) has electric heating elements (stove/oven, clothes dryer, coffee maker, iron, hot water heater, electric blanket, heated water bed), or (3) uses electric light bulbs. In addition to the 60 Hz fields associated with these household devices, many generate electromagnetic fields of various frequencies, especially when the appliance is energized. Fluorescent lights and light dimmer switches emit electromagnetic fields at other frequencies in addition to 60 Hz. Commonly used appliances such as television sets and computer terminals generate frequencies in the kHz range that expose children and adults from a few minutes to eight or more hours per day. Other sources of nonionizing radiation that seem logical to include in this chapter are devices that operate at microwave frequencies, for example, microwave ovens and anti-theft devices.

2.10.2 Distribution of Levels and Exposure

Biologically relevant exposures to electric and magnetic fields from power distribution systems, building wiring, and electric appliances are widespread. The most obvious exposure occurs in the electric fields beneath high-voltage transmission lines that carry electric power over long distances. The electric fields generated may be in the 1 to 2 kV/m^2 range. Less obvious are the magnetic fields emitted from the lower voltage distribution lines in front of most homes. These fields may be in the 1 to 2 mgauss range. Further, the electric and magnetic fields due to building wiring are of the order of 0.5 mgauss and 10 V/m, which can peak to over 10 times that amount when electrical equipment is energized. One obvious example of exposure to biologically-relevant fields is from electrically heated beds that are used for six to ten hours a night during the cooler months of the year. The 60 Hz electric field measured 30 cm from various electrical appliances range from 250 V/m^2 for the electric blanket to 2 V/m^2 for the incandescent light bulb. Electric fields at the center of rooms in a typical home range from 0.8 to 13 V/m^2 (Sheppard and Eisenbud, 1977).

It has been determined that man-made fields can be many orders of magnitude stronger than those of natural origin. However, many monitoring questions remain unanswered about the prevalence of man-made electric and magnetic fields normally found indoors, including their intensity, periodicity, and relative orientation with respect to naturally occurring magnetic and electric fields. All these aspects are relevant to potential biological reactions to the presence of these fields.

2.10.3 Health Effects of Nonionizing Radiation

The most prominent human health effects so far observed from exposure to electromagnetic fields, caused by the use of electric power, occur in the areas of cancer and reproduction. The experimental results that cause the most concern for human health come from two independent epidemiological studies, both of which arrive at the same conclusion, namely, that magnetic fields from distribution lines in the front of homes are positively correlated with an increased incidence of childhood cancer (Wertheimer and Leeper, 1979; Savitz et al., 1986). In another recent epidemiological study conducted in Sweden (Tomenius, 1986), an increased incidence of nervous system tumors was found in a population exposed to power-line frequency fields, in this case 50 Hz fields. This report introduces another area of concern, namely, the interaction of electromagnetic fields with the central nervous system.

Home appliances may be another area of health concern. Wertheimer and Leeper (1986) found the use of the electric blanket and the electrically heated water bed to be positively correlated with (1) increased spontaneous abortions for mothers exposed during the first trimester and (2) a reduction in birth weight and a lengthening of the gestation period for infants born to exposed mothers. In addition, there is laboratory research (see 6 below) showing that electric fields affect brain tissue from chicks exposed during embryogenesis, organogenesis, and organ maturation at exposure levels that are more than one order of magnitude below those experienced by the pregnant women studies. Another laboratory report indicates potential teratogenic effects from exposure to fields emitted from television sets and computer terminals. Thus some effect of ELF and higher frequency (kHz) fields on the fetus is possible.

Although no cause-and-effect relationship can be proven in the epidemiological studies, the number and mutually supportive results indicate that more detailed research is needed. There are laboratory results that are consistent with these epidemiological reports and that indicate the complexity of the interplay between the electric and magnetic fields and biological systems. Some of the more pertinent laboratory reports are cited below:

1. The recent result of a study by Phillips et al. (1986) indicates that human cells from certain tissues are susceptible to exposure from 60-Hz fields and can be altered in a way that enhances their transformation into the cancerous state.

2. Cells from the immune system can be affected by 60 Hz fields in a way that diminishes their natural function to seek out and destroy invading organisms. Luben et al. (1982) have shown that the cytotoxic activity of lymphocytes can be inhibited by electromagnetic field exposures. A weakened immune system would not be able to fully respond to the needs of an organism to fend off infections from outside the body and possibly prevent cells within the body from changing to a more cancerous state.

3. A classical biological system, the Dipteran salivary gland, has recently been shown to undergo unexpected changes in response to 60 Hz fields (Goodman and Henderson, 1986). These changes involve the induction of messenger RNA and the production of proteins from genes that were dormant. This demonstrates that 60 Hz fields can induce changes that affect fundamental biochemical pathways which, in some cases, could lead to the induction of oncogenes and ultimately to cancer.

4. Teratogenic effects were observed in the fetuses of mice exposed to a level and frequency of magnetic field commonly measured in the vicinity of television sets and computer terminals. A meeting presentation by Tribukait and colleagues (1986) from the Karolinska Institute has caused considerable scientific interest, but no other work has been reported that independently examines this result.

5. Brain tissue in vitro can be affected by 60 Hz fields at levels commonly found in homes. Blackman et al. (1985) have shown that certain frequencies of electric and magnetic fields can alter the interaction of calcium with brain tissue. Since calcium is important for neurotransmitter release and membrane integrity, and since it operates as a second messenger in biological systems, this perturbation may prove to have physiological significance.

6. A recent report at the Bioelectromagnetics Society Annual meeting (Blackman, 1986) indicated that the electric-field intensities found in homes could be biologically active in causing changes in the brains of developing organisms; these changes remained after the chicks were hatched. In this case, those animals exposed during gestation to the power-line frequency used in the United States were different from those exposed to the power-line frequency used in Europe. The consequences for humans are unknown, but may be associated with an increase in generalized stress or to some more specific ailment, either alone or in combination with other agents.

7. The earth's magnetic field was recently discovered to affect the particular frequencies of fields that cause biological changes. Blackman et al. (1986) showed that effective frequencies could be made ineffective, and vice versa, solely by making slight adjustments in the local value of the earth's magnetic field. This result has stimulated a number of hypotheses of the mechanism(s) of interaction and experimental research.

8. One such test of the influence of the earth's magnetic field showed that behavior in rodents could be altered by exposure to 60 Hz fields under selected, but widely prevalent, magnetic conditions (Thomas et al., 1986). The data (1) support the findings of Blackman and others that 60 Hz fields can cause changes in biological systems, and (2) show that exposure can eventually result in changes in the live animal, in this case, behavioral modification. The underlying mechanism has not yet been discovered, thus the consequences for human health are unknown.

In both the epidemiological reports and the laboratory experiments, some dose response data have been acquired. However, the results are not all internally consistent. Further, the average time for an effect to occur can vary widely depending on the initial conditions of the biological system, the ambient, natural electric and magnetic environments, the prior exposure history, and the presence of co-stressors. Thus, the results provide strong support for the need to establish the underlying mechanism(s) of action so that appropriate judgments can be rendered regarding relative risk from exposure.

2.10.4 Estimate of Population at Risk

The results of the epidemiology studies suggest two principal areas of concern for human health: reproduction and carcinogenesis. Thus the human fetus is potentially at risk. Furthermore, the epidemiological evidence suggests that human beings of all ages are potentially at risk for cancer. Laboratory data that support the latter suggestion show that exposure to electromagnetic fields may reduce the immune response, alter the utilization of genomic information, and stimulate biochemical activity involved with transduction of the genetic message. An epidemiological report and additional laboratory evidence demonstrate that exposure to ELF fields can induce changes in the nervous systems of animals. The changes include cancer, behavioral alterations, and biochemical changes. The latter two responses may add to a generalized increase in stressful living conditions for the entire population.

2.10.5 Mitigation and Control Options

There are a variety of options to limit exposure to electric and magnetic fields caused directly or indirectly by electric power usage. The particular methods to be selected would depend on understanding the mechanism of action so that safe limits can be ensured. For example, a change in the phasing of current flow in adjoining wires outside homes and in restructuring the return ground currents might be sufficient to eliminate the childhood cancer problem. Appropriate juxtaposition of wires in electric blankets and in water bed heaters could reduce fields from these sources. Fields from television sets and from computer terminals might be reduced by appropriate shielding and counter measures, such as a dummy fly-back transformer to emit the inverse wave form. Fields from these and other appliances such as light dimmers and microwave ovens may require individual mitigation techniques designed to reach certain limits defined by future health effect studies.

2.10.6 Conclusions

There are data consistent with a possible connection between exposure to power-line frequency electric and magnetic fields and 1) the appearance of cancer from basic biological changes in the utilization of genetic information (Goodman and Henderson, 1986), 2) to enhanced cellular transformations in test tubes (Phillips et al., 1986), 3) reduced ability to resist immunological challenges (Luben et al., 1982), 4) an increase in human childhood cancer (Wertheimer and Leeper, 1979). In another area, effects have been shown on brain tissue *in vitro* (Blackman et al., 1985), on brain tissue in animals exposed before they are born (Blackman, 1986), and on certain behaviors of animals (Thomas et al., 1986). Thus the specter of potential cancer induction/promotion and of aberrant behavioral changes have been associated with exposure to fields in homes caused by the electric power system and the use of home appliances. These reports do not unequivocally demonstrate that power-line frequency fields are a human health hazard separate and apart from the known hazard due to electric shock and burns; however, the combined reports do indicate that a great deal of caution must be exercised before allowing any increase in exposure of the general population until a better understanding is obtained of the underlying mechanism of action and possible synergism with other potentially hazardous agents and stressors in the environment.

There is a growing public awareness of the potential harm that may be caused by exposure to power-line frequency electric and magnetic fields. For example, the case of a 1985 settlement in Harris County, Texas which involved exposure of 4000 children in schools near a 345 kV transmission line. The jury felt there was a reasonable chance of hazard to the children's health. (Transmission/Distribution Health and Safety Report, 1986).

3. BUILDING SYSTEMS

3.1 INTRODUCTION

Previous sections of this report dealt with indoor air on a pollutant basis. Sources, effects, and IAQ control options were discussed on a pollutant category basis. In this section we will look at IAQ as a building related issue.

The interactions of sources of pollution and the building system determine IAQ. The building system, as used in this report, includes all aspects of the building. The structural aspects of the building, the arrangement of rooms and furnishings (including pollution sources), the construction materials used in the building, the movement of air from room to room via natural and mechanical ventilation, the heating, ventilating, and air conditioning system (HVAC) are all included in this definition. The term building system also covers the activities in the building.

The building system can:

- transport pollution (via either the HVAC or natural ventilation),
- remove pollution (e.g., forced exhaust to outside or natural exhaust by open windows or sink effects),
- introduce outside air pollution (e.g., via a ventilation inlet located near a loading dock), and
- control pollution (e.g., via air cleaners)
- produce pollution (e.g., off-gasing from materials), and
- affect ventilation (e.g., barriers can reduce ventilation).

The building environment (e.g., temperature, humidity, and lighting, affects the behavior of pollutants. For example, pollution emission rates are often a function of temperature. Interactions between pollutants are also affected by the environment. In some cases the effects of pollutants depend on environmental factors.

The building environment is especially important for biological pollutants. Temperature and humidity play a significant role in determining the types and quantities of biological pollutants that exist in a building (Morey et al., 1986).

Control of indoor air pollution requires careful operation of both sources and the building system. Sources should be removed from the building if possible. Sources that cannot be removed should be operated to minimize pollution. Pollution should be confined to areas near sources. Adequate ventilation throughout the building can prevent pollution build-up.

Although the building system plays a key role in determining IAQ, there has been little systematic work examining the role of the building. Much of the research has been ad hoc research related to a specific building.

3.2 THE BUILDING SYSTEM AS A SOURCE OF INDOOR AIR QUALITY PROBLEMS

3.2.1 General

The building system contributes to indoor air problems in a variety of ways. One of the most obvious ways that the building system affects IAQ is through air circulation and ventilation. Poor ventilation, low ventilation effectiveness, and transport of pollutants, are examples of air circulation effects.

The building system can be a source of pollution emissions. Emissions can be generated by materials and furnishings in the building or by the HVAC system. Other portions of the building system, for example, the plumbing system, can also generate air pollution emissions.

The arrangement of space, equipment, and activities in a building also affects IAQ. Walls, doors, and furnishings determine circulation patterns and affect ventilation effectiveness. The location of pollution generating activities in relation to other activities is important.

3.2.2 Ventilation Problems

Poor ventilation, which is one of the most obvious causes of poor IAQ, takes several forms, as described below.

- insufficient ventilation, where the amount of fresh air introduced into a building is inadequate
- ineffective ventilation where some portions of the building do not receive adequate ventilation
- inadequate exhaust of air, where the amount of air removed from an area of high emissions is insufficient to transport the pollutants (Wallingford and Carpenter, 1986; Ferahian, 1986; Seppanen, 1986)

The building system serves as a highway for moving pollutants from one area of the building to another. Q fever outbreaks, for example, are caused by transport of this infectious agent, a virus, through the HVAC system (Bayer, 1982).

Ventilation provides a pathway for polluted outdoor air to enter the building. An open window near a loading dock, for example, provides an easy way for pollutants generated by trucks at the dock to enter the building. An air intake vent located near a source of pollution provides a pathway for outside pollution to enter the building.

The arrangement of furnishings in the building can disrupt ventilation and air circulation. This often results in reduced ventilation effectiveness and poor IAQ.

3.2.3 Source Effects

The building system provides breeding grounds for many biological contaminants. The dust and other materials that collect in ventilation duct work often provide an ideal medium for the growth of various biological contaminants (Morey et al., 1986). Fiberglass duct work, as it ages and collects a variety of organic material on its roughened surface and in crevices formed by glass fiber mats, becomes an excellent growth medium for molds, under appropriately humid conditions. Fiberglass batting used to line duct work to reduce noise acts as a filter for dust and other particles, which provide good media for the growth of molds and/or bacteria. The duct work also provides a pathway for distributing the biological contaminants throughout the building.

Biological contaminants can also grow on furniture and furnishings (Morey et al., 1986).

Gaseous organic pollution can be produced by off-gassing from plastic duct work. Fibers can be generated by abrasion of duct material, and dust and other materials collected in a duct may be resuspended from time to time. A cycle of pollutant collection followed by resuspension, for particles, or regeneration, for gases, can then be established.

Materials such as minerals and conditioning additives from water used in humidification systems may also be spread throughout the building (Burge, 1985). Some types of electrostatic air cleaners may generate ozone when improperly operated. Dirty air filters can also reduce IAQ (Kress and Fink, 1986). Other portions of the building system such as the plumbing system, swimming pools, and construction materials also affect IAQ (Hodgson and Kreiss, 1986; Gamble et al., 1986; Morey et al., 1984; Wallingford and Carpenter, 1986). As discussed in other sections of this report, the materials used in the building can be major sources of pollutants. Once these pollutants are released, they can be then transported throughout the building.

Pollutant interactions can occur within buildings. For example, organics and bioagents can adsorb to particles, which are then transported throughout the building. Other important interactions include particle formation from condensation and particle evaporation. These processes may occur in the building space or in the HVAC system. Vaporization of organic vapors from tobacco particles collected by air cleaners can be a significant problem in situations where air cleaners are being used to provide "smoke-free" air.

3.2.4 Arrangement of Building Space and Activities

The arrangement of space, doors, walls, and furnishing is important for IAQ. Walls, partitions, and furnishings affect air circulation and ventilation. Furnishing can be located to block air intakes or air outlets and thus reduce ventilation effectiveness.

Quite often the original design of a building is based on assumptions of where specific activities will occur. Activity patterns may be changed, and pollution producing activities moved into new locations. New activities and equipment may also be introduced into the building. Unless care is taken in locating these activities and equipment, an IAQ problem may be created.

Location of people and their activities is of growing importance. This is especially true with regard to smoking. Many local regulations require that smoke-free air be provided. Providing such air while allowing smoking will require careful attention to the location of smokers in a building.

3.3 MITIGATION OF INDOOR AIR POLLUTION

3.3.1 General

The building system can play a major role in reducing indoor air pollution. A properly designed and operated building can eliminate or reduce indoor air pollution by:

- confining pollution to areas near sources,
- exhausting pollution before it mixes with building air,
- diluting pollution with outside air, and
- removing pollution with air cleaners.

Indoor air pollution control requires combining source control with good building system operation. Source control minimizes the amount of pollution generated. Building system operation can then minimize the impact of the pollutants generated. Details on source control are presented in previous sections of this report.

A building system approach to IAQ mitigation will result in the most cost-effective mitigation. Under the building system approach, all aspects of the problem and control options are analyzed. The effects of various actions on each other are also analyzed, as are the economics of each option. Finally, the most cost-effective option is selected.

Because many of the techniques used for IAQ control are discussed in other sections of this report, most of the discussion in this section will be brief.

3.3.2 Confining Pollution

Confining pollution-producing activities to specific locations is a cost-effective way of improving IAQ. For confinement efforts to be effective, the air circulation between the pollution locations and the rest of the building must be minimized. Thus, separate HVAC systems should be provided for areas which generate pollution.

3.3.3 Exhausting Pollution

Exhausting pollution before it mixes with the rest of the building is another common mitigation procedure. Cooking stoves, for example, may have hoods and filters to remove pollutants before they can mix with the building air. Bathroom moisture and odors can be exhausted directly to keep them from the rest of the house. Exhausting pollution can be combined with confining pollution in a effective manner.

Local exhaust of pollutants can be a cost effective method of improving IAQ. This technique is often overlooked. Local air cleaning is another option that can be used in many situations.

3.3.4 Dilution With Outside Air

Many IAQ problems can be mitigated by increasing building ventilation. The increase in ventilation reduces pollutant concentrations by dilution with outside air. However, modern practices of indoor climate control may make increased ventilation economically unattractive. Increasing the ventilation rate increases the amount of air to be heated or cooled, and increases the amount of treated air that is exhausted to the outside. While increasing ventilation is a theoretical possibility for improving IAQ when outdoor air is of satisfactory quality, it may not always be practical. Increased ventilation effectiveness may eliminate or reduce the need for increased ventilation (Seppanen, 1986). An increase in ventilation effectiveness makes maximum use of the building ventilation.

3.3.5 Air Cleaners

Air cleaning is one way to gain some of the benefits of increased ventilation without diluting with outside air. High efficiency air cleaners can remove particulate matter and other pollutants. The major drawbacks of most of the currently available air cleaners are low efficiency for many pollutants, high capital costs, and high operating costs. Improperly operated and maintained air cleaners can even increase pollutant levels.

The efficiency of many types of particulate air cleaners is not well known. This is especially true for removal of small particles which are important for defining health effects. The performance of air cleaners for gas, especially at the low concentrations found indoors, is also largely unknown.

Air cleaner effectiveness is determined by a combination of the efficiency of the air cleaner and the rate at which air is circulated through the cleaner. High effectiveness requires high air circulation rates (several changes per hour) and avoidance of short-circuiting (returning the air cleaner exhaust directly to its intake).

Air cleaners can help mitigate a wide range of IAQ problems, for example, ETS levels can be reduced with high efficiency air cleaners.

3.3.6 Selection of Materials

As discussed in previous sections of this report, the materials used in a building can be major sources of pollution. Proper selection of materials can improve IAQ.

3.3.7 Elimination of Entry Routes

In cases where the IAQ problem is caused by entry of pollutants from outside the building, entry routes should be eliminated. The actions required to eliminate the entry routes depend on the nature of the problem. For example, pollutants entering due to poorly placed air intakes can be blocked by relocating the air intake.

See the radon (Section 2.7) and pesticides (Section 2.9) discussions for other techniques for eliminating entry routes.

3.4 COMFORT AND OTHER ISSUES

Comfort is an important part of IAQ. Mitigation measures must address comfort, as failure to do so can reduce or eliminate the benefits of the mitigation effort.

There is a danger that comfort will be reduced when increased ventilation or increased air circulation are used to improve IAQ. High ventilation or circulation rates may create drafts that cause discomfort. As a result people may block air circulation ducts to aleviate their discomfort and thus eliminate many of the benefits for high ventilation rates. Because drafty rooms are perceived as cold, people may also turn up the thermostat to increase heat, which imposes additional energy penalties.

Building climate (temperature and humidity) can be controlled to minimize IAQ problems. Control of temperature and humidity is especially important for controlling biological pollutants such as molds. Many biological pollutants require special temperature and humidity conditions in order to grow.

Odors are another comfort-related issue. Ventilation standards are based in part on odor impressions (American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., 1981). Odor is an important component of ETS and the perception of smoke-free air (Clausen et al., 1986a,b).

The general importance of comfort is addressed by Davidge (1986), who discusses the interactions between various factors affecting comfort. Individual comfort factors may be judged satisfactory, but their interactions may result in the perception of poor air quality.

3.5 MEASUREMENT AND DIAGNOSIS OF BUILDING SYSTEM FACTORS

3.5.1 General

Solving IAQ problems requires careful diagnosis to identify problems and solutions. Diagnosis is often hindered by the large number of potential pollutants (Berglund et al., 1986). The identification of individual pollutants is very difficult, especially since the problem may not be due to any single pollutant. Some progress has been made in understanding the interactions between pollutants, but more work is needed (Berglund et al., 1986).

Tracing a pollutant to a source is also often difficult. Monitoring techniques that improve source identification are lacking. Modeling is necessary to better understand the effects of the building system on IAQ and it is useful in determining the effects of IAQ on health (Leaderer, 1986).

3.5.2 Air Circulation/Ventilation

Air circulation in a building is a combination of natural and mechanical circulation. The air circulation in most commercial buildings is dominated by mechanical circulation. Mechanical circulation is also important in many homes for much of the year.

Measurement of air circulation falls into three main categories:

a. Measurement of air circulation.

b. Measurement of total ventilation (mixing with outside air).

c. Measurement of local ventilation.

Air circulation measurements can be made in HVAC ducts or in the various parts of the building. The measurements are made with instruments designed for low flow rates.

There are a variety of methods for measuring ventilation (air exchange with outside). Methods for measuring overall ventilation are fairly simple, inexpensive, and reliable. Methods for determining ventilation of individual rooms are expensive. Some of the more common methods are summarized in Table 3-1.

Methods for relating the various measurements of air circulation and ventilation to ventilation effectiveness are not well developed.

3.5.3 Pollutant Concentrations and Identification

Indoor air is a complex mixture made up of a host of chemicals. The chemicals may be present as particles or gases. Quite often the gases can be sorbed onto the particles. The analysis of this complex mixture is difficult and requires a variety of instruments.

3.5.3.1 Particles. The particles in indoor air range in diameter from less than 0.05 to greater than 100 micrometers. The chemical composition of the particles ranges from simple inorganic compounds to complex organic compounds. The particles may be of a biological nature such as pollens or molds, bacteria or viruses, or they may be non-biological particles that are associated with allergenic molecules of biologic origin.

Analysis of the particulate problem in a building starts with the analysis of the particle size distribution throughout the building. The instruments necessary for these measurements are discussed in the particulate section of this report. The particle size distribution is the most important physical property of the particles in a building. Most of the behavior of the particles, such as transport, interactions with other pollutants, and deposition in lungs depends on the particle size distribution.

Techniques discussed in other sections of this report can be used to determine the chemical composition of the particles. Biological activity of the particles should also be determined.

3.5.3.2 Gases. Analysis of the gases found in buildings is discussed in the various pollutant sections in Chapter 2. Because indoor air is a complex mixture, these measurements can be quite difficult and expensive. In cases in which the identity of the pollutant is known, the expense and difficulty can be reduced by limiting measurement to the problem pollutant. Unfortunately, in most cases the pollutants causing specific problems are not known and a full-spectrum analysis is necessary (Hedge et al., 1986).

TABLE 3-1. METHODS OF AIR INFILTRATION MEASUREMENT

Method	Determines	Advantages		
Tracer gas (instantaneous)	Ventilation of who or part or part of building	Gives information about ventilation rate under running conditions.	Indirect method. Result depends on actual conditions. Mixing is difficult. Needs special training.	Simultaneous air changes/hr
Tracer gas (continuous)	Ventilation of whole or part of building	Gives ventilation rate over long time under different conditions.	Indirect method. Expensive.	Air changes at operating conditions
Tracer gas container sampling	Ventilation of whole or part of building	Simple and inexpensive.	Indirect method. Low control of taking samples.	
Whole house pressurization	Airtightness of the building building envelope	Gives information about the leakiness of the building envelope. At high pressure differences the method is relatively insensitive to weather. Inexpensive.	No information about actual ventilation degree.	Air leakage at high pressured differences
Component pressurization	Airtightness of the building envelope	Quantifies air leakage through building components. Simple equipment.	Time to adjust equipment.	Air leakage
Thermography	Qualitative detection of leakage sites	Provides information about leakage sites and defects in the thermal insulation.	Expensive. Needs temperature difference of at least 10°C. Requires special training.	Leakage sites
Smoke Pencil	Qualitative detection of leakage sites	Provides Information about leakage sites and air movements. Simple and inexpensive.	Difficult to find leaks, especially with internal pressurization.	

3.5.4 Questionnaires

Conventional measurements of pollutant concentrations often fail to uncover IAQ problems. In these situations, questionnaires are often the only tool for defining the problem (Hedge et al., 1986). In most cases properly designed questionnaires play a key role in diagnosis of building problems. Follow-up questionnaires are used to determine whether or not corrective actions have solved the problem. The value of a questionnaire is strongly dependent on its design and on the analysis of the responses.

3.5.5 Diagnosis

The purpose of making the measurements discussed above is to diagnose a building problem. The diagnosis includes identification of pollutants causing the problem, identification of the sources of pollution, and identification of building related factors that allow the pollutant to be a problem. When the diagnosis is complete, action to mitigate problems can be be implemented.

Relating the measurements to problems and sources is a difficult job. Although there are case-study examples of the diagnosis process, a systematic diagnosis procedure has not been developed. Procedure development is hindered by the fact that indoor air is a complex mixture problem. Quite often there is no single element or simple combination of elements that separates a problem building from a nonproblem building (Hedge et al., 1986). Even when the factors causing the problem are identified, tracing these factors to a source can be difficult. This process could be improved if signatures for various sources could be determined.

There are cases where diagnosis has been successful (Hodgson and Kreiss, 1986). In many cases, however, the exact cause of a problem remains unknown. In these situations it is easier to recommend increased ventilation as a mitigation approach than it is to fully diagnose the problem.

3.6 INDOOR AIR MODELS

Analysis of pollutants and buildings is necessary for understanding IAQ. Well-integrated IAQ models are essential for this analysis. The IAQ models should include all aspects of the building system, including sources and sinks for pollutants, pollutant interactions, ventilation, air cleaners, and health effects, among others.

Some aspects of the IAQ problem have been well-modeled (Walton, 1985; Versar, 1987; McNall et al., 1985). Most of the available models concentrate on the ventilation aspects of indoor air. These ventilation models are useful to those involved in designing ventilation systems. Because of the interest in energy conservation, models for analysis of energy impacts have been developed. The U.S. Department of Energy continues to develop such energy impact models. The available models are research models that describe limited parts of the IAQ problems in detail. The models have not been developed to the level of usefulness or ease of use required for general application (Versar, 1987). While the available models provide an analysis of the specific process being modeled, they neglect important parts of the indoor air problem. Sources and sinks for pollutants, pollutant interactions, and air cleaners are a few of the areas neglected by most IAQ models.

Realistic representation of source and sink terms is essential to understanding IAQ. Important data necessary to model source and sink terms realistically are now being developed through EPA's IAQ program.

Present models tend to neglect the factor of pollutant interactions, which often determine how pollutant levels will affect people. For example, many gaseous pollutants (organic and inorganic) adsorb to particles that carry the pollutant to the lung. In the absence of particles, the gaseous pollutants might not reach the lung. The interaction of radon and particulate matter is an example of such a carrier phenomenon.

Little work has been done to include the effects of realistic air cleaner performance on IAQ in existing models since the effects of air cleaner operation on IAQ have not been developed. Models of particulate air cleaners do not include effects of particle diameter on air cleaner performance, yet this is critical to analyzing the effects of particulate air cleaners on IAQ.

Available models are also weak in correlating the effects on health or welfare of pollutants with the pollutant concentrations predicted by the model. Such integration in necessary if one is to estimate exposure reduction as a result of proposed mitigation efforts. When such models are developed they can be used to evaluate risks involved in a given indoor situation, and can be used to evaluate risk reduction offered by various mitigation options.

4. WELFARE CONCERNS

4.1 INTRODUCTION

Discussions in the previous chapters have focused on aspects relevant to the evaluation of health effects. To some extent, these considerations are similar to welfare effect assessment. As noted below, however, there are data requirements specific to materials damage, soiling, and odors. More important, however, is the identification of significant exposure scenarios. Of concern are situations in which materials susceptible to indoor air pollutant damage must perform an essential function or are valuable in cultural or historical terms. A frequently discussed example of the former case is telephone switching equipment, vulnerable to corrosive attack both by gases and soluble ions in airborne particulate matter (Sinclair and Psota-Kelly, 1985).

As a relatively young nation, the United States has had less occasion than its European counterparts to be concerned with preserving archives and cultural properties from atmospheric attack, either in outdoor or indoor exposures. A report titled Impact of Air Pollutants on Materials, developed for the North Atlantic Treaty Organization's Committee on the Challenges of Modern Society, highlighted the member nations' concern for effects on cultural property. Results of investigations documenting observed damage to objects housed in museums, galleries, archives, and similar structures were reported. A number of protective and remedial techniques were discussed.

The U.S. approach to estimating welfare effects of materials damage and soiling has been almost entirely focused on estimates of cost of damage to materials exposed to ambient air pollution. For soiling, the perspective has often been limited to residential or household cleaning. Valuation of aesthetic or historic worth does not lend itself to development of variables for use in applying physical or economic damage functions. There is, for example, substantial evidence that "indoor soiling causes significant and, in some cases, irreversible damage to cultural property" (Baer and Banks, 1985). Dust, soot, ETS residue, textile fibers and alkaline aerosols from setting concrete have all been encountered in the deterioration of works of art, documents, and historic artifacts. Paper, paintings, textiles, and wax objects have been identified as especially susceptible to particulate pollution; metals and photographic materials are vulnerable to attack by acid fumes. Protective measures such as special air conditioning and filtering systems have been developed to mitigate these effects on works of art and other cultural properties in museums, galleries, and libraries.

The questions to be answered regarding indoor air pollution and materials damage, soiling, and odors relate to the need for identification of incremental effect: In what situations will adverse welfare effects attributable to indoor air pollution be an important factor relative to other factors commonly encountered? To what extent, for example, is the decision to replace, repair, or take mitigative action solely a function of indoor air pollution? With regard to soiling of indoor surfaces, is the decision to clean a function of particle accumulation, or are other considerations (e.g., regular cleaning schedule, volume and nature of activity) of equal or greater significance? Are materials damaged at a rate significantly greater by air pollutants than by normal wear and tear? How much is variability in perceived odor a function of pollutant concentration, and how much a function of receptor sensitivity?

As noted above, research on the effects of air pollutants on materials has focused on exposures to ambient air concentrations. Where exposures are similar in the indoor environment, the research data so developed are applicable to materials exposed to indoor air pollutants. As Baer and Banks (1985) note, institutions with air conditioning systems include some filtration for particulate matter, and often are equipped to remove SO_2 and/or ozone. Oxides of nitrogen do not appear to be significantly attenuated by building ventilation systems (National Research Council, 1986b). Nazaroff and Cass (1986), for example, found levels of NO and NO_2 in a California museum gallery to be essentially the same as outdoors. The following summary, excerpted from previous reviews, gives a general description of the effects of air pollution on materials that may be found in association with indoor exposures.

Table 4-1 summarizes the types of damage that can be caused by indoor exposure to air pollution. The important points to be brought out from Table 4-1 are that a variety of materials are damaged by common air pollutants, that sulfur oxides figure prominently as a pollutant type that can cause damage to

TABLE 4-1. AIR POLLUTION EFFECTS ON MATERIALS

Materials	Type of Damage	Principal Air Pollutants	Other Environmental Factors
Metals	Corrosion, tarnishing	Sulfur oxides and other acid gases	Moisture, air, salt, microorganisms, particulate matter
Paint and organic coatings	Surface erosion, discoloration, soiling	Sulfur oxides, hydrogen sulfide, particulate matter	Moisture, sunlight, ozone, microorganisms
Textiles	Reduced tensile strength, soiling	Sulfur oxides, nitrogen oxides, particulate matter	Moisture, sunlight, ozone, physical wear
Textile dyes	Fading, color change	Nitrogen oxides, ozone	Sunlight
Paper	Embrittlement, soiling	Sulfur oxides, particulate matter	Moisture, physical wear
Magnetic storage media	Loss of signal	Particulate matter	Moisture, heat, wear
Photographic materials	Microblemishes, "sulfiding"	Sulfur oxides, hydrogen sulfide	Moisture, sunlight, heat, other acid gases, particulate matter, ozone and other oxidants
Rubber	Cracking	Ozone	Sunlight, physical wear
Leather	Weakening, powdered surface	Sulfur oxides	Physical wear
Ceramics	Changes surface appearance	Acid gases, HF	Moisture, microorganisms

Source: Yocom et al. (1982), Baer and Banks (1985), National Research Council (1986b), Yocom et al. (1986), Murray et al. (1986).

many materials, and that air pollution is only one of the several factors that can cause damage to materials exposed to indoor atmospheres. In order to adapt research results produced for other purposes to an assessment of indoor air's effects on materials, soiling, or odors, several data requirements specific to the evaluation of welfare effects must be considered.

Air quality measurement techniques designed for use in air pollution compliance monitoring are not likely to provide the information most useful to the assessment of effects on materials, because deposition measurements on surfaces are more relevant. For instance, the use of sampling equipment designed to represent the experience of the human lung inhaling air cannot also represent the experience of a surface passively exposed to polluted atmospheres. Sampling techniques designed to model inhalation could, however, be appropriate for data gathered in connection with an assessment of malodorous pollutants.

Other aspects of data requirements in which welfare effect considerations vary markedly from those of health effects have to do with the need for measurements of such parameters as relative and/or absolute humidity and temperature. Not only can deposition be strongly affected by such factors, but most important effects on materials will not occur significantly except under certain surface conditions. For example, surface moisture is a necessary prerequisite for significant metal corrosion. The algorithm for exposure, then, is not usually limited to concentration times time, but surface concentration times the periods of time during which certain physical conditions have been met. The recent introduction of electrochemical sensors has facilitated meeting these data requirements in studies of metal corrosion, since they can provide a continuous documentation of corrosion that can be compared with time-dependent parameters (e.g., relative humidity, temperature, pollutant concentration) recorded at the same site (Mansfeld, 1982).

The same conditions that accelerate material degradation by air pollution also favor growth of biological agents that degrade and discolor, such as bacteria and fungi (Solomon and Burge, 1984). The effects of the action of these agents and of the air pollutants that are simultaneously present in an indoor environment may be very difficult to separate.

The lack of any absolute threshold concentration associated with materials damage, soiling, or odor is another important concept. In the case of materials damage, not only must certain environmental conditions be met, but a critical damage point must be reached before welfare can actually be said to be adversely affected. Perception thresholds are critical to assessing the impact of either soiling or odors. There is reason to believe that conditions specific to indoor spaces may well affect these perceptions (e.g., Engen, 1986; Clausen et al., 1986a,b).

4.2 MATERIALS DAMAGE

4.2.1 Introduction

Several fundamental problems in quantifying the extent of damage to materials from specific pollutants whether exposed indoors or outdoors are listed below.

- Many types of damage associated with air pollutants (e.g., corrosion, erosion, fading) tend to occur also in unpolluted atmospheres and therefore cannot be distinguished from those caused or enhanced by the presence of air pollutants. Thus, determining the isolated influence of a given pollutant is not a straightforward process.
- Laboratory studies in which individual pollutants are introduced to susceptible materials in known exposures independent of other influences tend to involve unrealistically high concentrations and otherwise are not representative of real-life outdoor exposure situations. This problem may be especially magnified relative to indoor exposures, where, according to Nazaroff and Cass (1986) "homogenous chemical reactions play an important role in determining pollutant concentrations."
- Changes made during recent years in the formulations of materials and protective coatings as a result of technological improvements can have a fundamental effect upon the nature and extent of air pollution-induced damage to materials and associated damage estimates.
- Determination of the quantities of materials in place in relation to air pollutant exposures is a difficult task, especially when data for significant materials (e.g., fabrics, paints and paper) exposed to indoor environments may not have been collected in this context.
- Data on deposition velocities for gases and particles to indoor surfaces are quite limited. Available information indicates that extrapolation from outdoor experience will not be valid (Nazaroff and Cass, 1986).

The air quality criteria documents for particulate matter and sulfur oxides (U.S. Environmental Protection Agency, 1982a), nitrogen oxides (U.S. Environmental Protection Agency, 1982b) and ozone (U.S. Environmental Protection Agency, 1986a) contain thorough reviews of the literature on material effects related to each of the pollutant classes. This chapter will rely heavily upon the parts of those documents that deal with effects on materials and soiling.

In a recent review of factors contributing to degradation of materials in the atmosphere, Graedel and McGill (1986) identified a number of air pollutants that may be present in indoor environments at levels as high or higher than those outdoors: NO_2, HCHO, formic acid (HCOOH), SO_2, and ozone (O_3). HCHO and HCOOH are of concern principally because of their effects on exposed metals. Principal effects and levels associated with NO_x, SO_x, and O_3, as identified in the EPA's air quality criteria documents for these pollutants, are summarized below.

4.2.2 Oxides of Nitrogen

The damaging effects of atmospheric oxides of nitrogen have been established for a variety of materials, including dyes, fibers, plastics, rubber, and metals. Field exposures of cotton, viscose rayon, cellulose acetate, and nylon fabrics colored with representative dyes demonstrate that fading occurs for specific dyes in air containing NO_2, O_3, and SO_2. These exposures were carried out in ambient air and protected against sunlight. Chamber studies using individual pollutants, NO_2, O_3, and SO_2, have shown that some individual dye-fiber combinations exhibit color fading only in response to NO_2 exposure, whereas others are susceptible to O_3, as well as combinations of NO_2 and O_3. SO_2 introduced an accelerating effect. Disperse dyes used for cellulose acetate and rayon include vulnerable anthraquinone blues and reds. The cellulosic fibers, cotton and viscose rayon, dyed with certain widely used direct dyes, vat dyes, and fiber reactive dyes, suffer severe fading on chamber exposures to 940 $\mu g/m^3$ (0.5 ppm) NO_2 under high humidity (90 percent) and high temperature (32°F) conditions. Significant fading is observed on 12 weeks exposure to 94 $\mu g/m^3$ (0.05 ppm) NO_2 under high humidity and temperature conditions (90 percent, 32°F).

Acid dyes on nylon fade on exposure to NO_2 at levels as low as 188 $\mu g/m^3$ (0.1 ppm) under similar conditions. Dyed polyester fabrics are highly resistant to NO_2-induced fading. However, permanent press fabrics of polyester cotton and textured polyester exhibited unexpected fading when first marketed. The fading was in the disperse dye, which migrated under high-heat conditions of curing or heat-setting to the reactive medium of resins and other surface additives.

The yellowing of white fabrics is documented for polyurethane-segmented fibers (Lycra and Spandex), rubberized cotton, optically brightened acetate, and nylon. Yellowing is also reported on fabrics finished with softeners or antistatic agents. NO_2 was demonstrated to be the pollutant responsible for color change, while O_3 and SO_2 showed no effect. Chamber studies using NO_2 concentrations of 376 $\mu g/m^3$ (0.2 ppm) for 8 hours showed yellowing equivalent to that on garments returned to manufacturers.

The tensile strength of fabrics may be adversely affected by the hydrolytic action of acid aerosols. NO_2 has been demonstrated to oxidize the terminal amine group ($-NH_2$) of nylon to the degree that the fiber has less affinity for acid-type dyes. Nylon 66 may suffer chain scission when exposed to 1,800 to 9,400 $\mu g/m^3$ (1.0 to 5.0 ppm) NO_2. Field exposures of fibers have emphasized the action of acids derived from SO_2, although NO_2 may also have been present in high concentrations in urban sites and may well be present at high levels indoors. Information on the contribution of NO_2 to degradation is incomplete.

4.2.3 Sulfur Oxides

Though physical damage functions have been developed for the effect of SO_2 on a number of materials, even the most reliable damage functions must be used with caution. More data are required to take into account orientation, location, and design of materials in use. Those damage functions listed in Table 4-2 were selected on the basis of their treatment of independent variables and their inclusion in major literature reviews. Damage functions vary in form, reflecting different parameters measured and methods of measurement. Time-of-wetness (often expressed as relative humidity above a critical value) is the most important variable in these damage functions.

Functions for zinc (Zn) or galvanized steel appear to show the best fit, followed by the functions for oil-based house paint. The field studies by Haynie and Upham (1970) and Haynie (1980) and the chamber study by Haynie et al. (1976) incorporated critical variables and provided relatively reliable damage functions for galvanized steel. The functions selected for weathering and enameling steel and for oil-based paint also utilized these critical environmental variables.

While the relationships between SO_2 exposure and effect are less well established, effects of sulfur oxides on other materials, including paint, fabrics, paper, and leather have been identified in the literature. These effects are of particular concern in the indoor environment when cultural properties such as archives and works of art are exposed to pollutants.

TABLE 4-2. SELECTED PHYSICAL DAMAGE FUNCTIONS RELATED TO SO_2 EXPOSURE

Material	Reference	Exposure-Response Relationships	R^2
Zinc	Haynie and Upham (1970)	$Y = 0.001028 (RH - 48.8) SO_2$	0.92
Galvanized steel	Haynie et al. (1976)	$corr = (0.0187 SO_2 + e^{41.85 - 23,240/RT}) t_w$	0.91
Galvanized steel	Haynie (1980)	$corr = 2.32 t_w + 0.0134 v^{0.781} SO_2 t_w$	Not provided by author
Oil-based house paint	Spence et al. (1975)	$Y = 14.3 + 0.0151 SO_2 + 0.388 RH$	0.61
Enameling steel	Haynie and Upham (1974)	$corr = 325 \sqrt{t}\, e^{[0.00275 SO_2 - (163.2/RH)]}$	Not provided by authors
Weathering steel	Haynie et al. (1976)	$corr = [5.64 \sqrt{SO_2} + e^{(55.44 - 31,150/RT)}] \sqrt{f_w}$	0.91

corr = depth of corrosion or erosion, μm

Y = corrosion/erosion rate, μg/yr

SO_2 = μg/m^3 SO_2

R = gas constant (1.9872 cal/gm mol K)

RH = percent annual average relative humidity

f_w = fractional time of panel wetness

t_w = time of wetness in years

v = wind velocity in m/s

T = geometric mean temperature of panels when wet, K

t = time exposure, years

Note: 1 ppm SO_2 = 2620 μg/m^3.

Source: U.S. Environmental Protection Agency (1982a).

4.2.4 Ozone

More than two decades of research show that O_3 damages certain nonbiological materials; the amount of damage to actual in-use materials, however, is poorly characterized. Knowledge of I/O O_3 gradients, though expanded considerably in recent years, has not been incorporated in materials damage studies. Moreover, virtually all materials research on photochemical oxidants has focused on O_3.

The materials most studied in O_3 research are elastomers and textile fibers and dyes. Natural rubber and synthetic polymers of butadiene, isoprene, and styrene, used in products like automobile tires and protective outdoor electrical coverings, account for most of the elastomer production in the United States. Little exposure to indoor environments is expected, then, for these materials.

The effects of O_3 on dyes have been known for nearly three decades. In 1955, Salvin and Walker exposed certain red and blue anthraquinone dyes to a 0.1 ppm concentration of O_3 and noted fading, which until that time was thought to be caused by NO_2. Subsequent work by Schmitt (1960, 1962) confirmed the fading action of O_3 and the importance of relative humidity in the absorption and reaction of O_3 with vulnerable dyes. The acceleration in fading of certain dyes by high relative humidity was noted later by Beloin (1972, 1973) at an O_3 concentration of 0.05 ppm and relative humidity of 90 percent. Kamath et al. (1982) also found that a slight rise in relative humidity (85 to 90 percent) caused a 20-percent dye loss in nylon fibers.

Both the type of dye and the material in which it is incorporated are important factors in a fabric's resistance to O_3. Haynie et al. (1976) and Upham et al. (1976) found no effects from O_3 concentrations of 0.1 to 0.5 ppm for 250 to 1000 hr under high and low relative humidity (90 versus 50 percent) on royal blue rayon-acetate, red rayon-acetate, or plum cotton. On the other hand, Haylock and Rush (1976, 1978) showed that anthraquinone dyes on nylon fibers were sensitive to fading from O_3 at a concentration of 0.2 ppm at 70 percent relative humidity and 40°C for 16 hr. Moreover, the same degree of fading occurred in only 4 hr at 90 percent relative humidity. At higher concentrations, there was a parallel increase in fading. Along with Heuvel et al. (1978) and Salvin (1969), Haylock and Rush (1976, 1978) noted the importance of surface area in relation to the degree of fading. In explaining this relationship, Kamath et al. (1982) found that O_3 penetrated the fiber itself and caused most of the fading through subsequent diffusion to the surface.

Field studies by Nipe (1981) and laboratory work by Kamath et al. (1982) showed a positive association between O_3 levels and dye fading of nylon materials at an O_3 concentration of 0.2 ppm and various relative humidities. In summary, dye fading is a complex function of O_3 concentration, relative humidity, and the presence of other gaseous pollutants. At present, the available research is insufficient to quantify the amount of material damage attributable to O_3 alone. Anthraquinone dyes incorporated into cotton and nylon fibers appear to be the most sensitive to O_3 damage.

The degradation of fibers from exposure to O_3 is poorly characterized. In general, most synthetic fibers like modacrylic and polyester are relatively resistant, whereas cotton, nylon, and acrylic fibers have greater but varying sensitivities to the gas. O_3 reduces the breaking strength of these fibers, and the degree of reduction depends on the amount of moisture present. Under laboratory conditions, Bogaty et al. (1952) found a 20 percent loss in breaking strength in cotton textiles under high-moisture conditions after exposure to a 0.06 ppm concentration of O_3 for 50 days; they equated these conditions to a 500- to 600-day exposure under natural conditions. Kerr at al. (1969) found a net loss of 9 percent in breaking strength of moist cotton fibers exposed to O_3 at a concentration of 1.0 ppm for 60 days. The limited research in this area indicates that O_3 in ambient air may have a minimal effect on textile fibers, but additional research is needed to verify this conclusion.

The effects of ozone on paint are small in comparison with those of other factors. Some studies (Yocom et al., 1986) of air pollutant effects on cultural properties indicate that ozone may fade or alter pigments used in watercolors and Japanese prints.

4.2.5 Particulate Matter

Though the most significant welfare effects of particles suspended in indoor air are associated with soiling, (see Table 4-1 and Section 4.3, below), there is considerable evidence that water-soluble salts have potential impact in areas where electronic equipment is located. As electronic equipment is becoming more common in both office environments and in homes, the significance of this effect will be greater than at present. The electronics industry has a special concern for these aerosol components, as hygroscopic elements of suspended particulate matter can lead to corrosion, current leakage, and subsequent failure of electronic equipment. In reporting the results of a study to characterize the water-soluble components of size-fractionated aerosols collected in an office building, Walker and Weschler (1980) suggest that when electronic equipment is involved, the relative humidity of the indoor space should be controlled at a level consistent with the deliquescent properties of the salts.

4.3 SOILING

The previous discussion has dealt with the effects of air pollutants on materials. In distinguishing between materials damage and soiling, we may say that materials damage occurs when particle accumulation alters a surface in a way that is not reversible by a cleaning operation. When the surface alteration can be undone by cleaning, soiling has occurred. A flow chart for analysis of factors contributing to soiling welfare effects is depicted in Figure 4-1. Airborne particulate matter is deposited at some rate on a surface. The resulting particle accumulation produces a physical effect that can be measured by instrumental means. This physical effect at some level provokes a visual response. At some level, the visual response is characterized as offensive. This aesthetic effect at some level provokes a behavioral response either to remove the offending particles from the affected surface, or to accept the visual insult. The result of both behavioral responses--either utility lost or resources expended--may be assigned a dollar value. The magnitude of the economic effect depends upon the activity necessary to remove the actual offense.

To evaluate soiling as a welfare effect of airborne particles, the relationship of airborne particles to physical effect and the physical effect's relationship to the sensory effect must be established. Table 4-3 illustrates the relationships between airborne particles and the various stages of particle surface effects associated with soiling, as developed in literature cited in the criteria document for sulfur oxides and particulate matter (U.S. Environmental Protection Agency, 1982a). Thus, the vertical columns labeled physical, sensory, aesthetic, and economic effects show the kind of consideration given by the cited study to that particle surface effect. It is clear that no single study has considered all of these effects and their interrelationships.

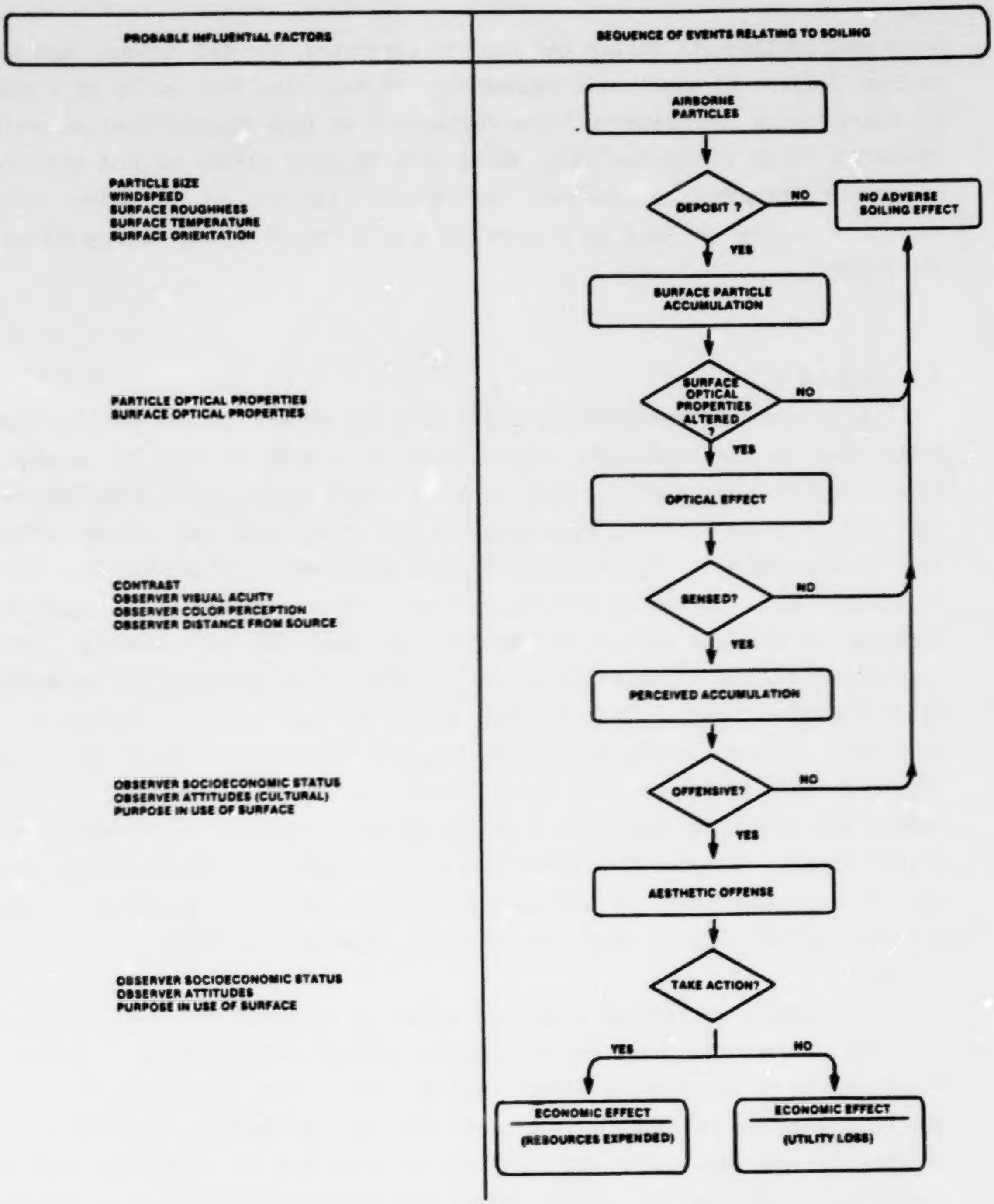

PROBABLE INFLUENTIAL FACTORS
SEQUENCE OF EVENTS RELATING TO SOILING
AIRBORNE PARTICLES
PARTICLE SIZE
WINDSPEED
SURFACE ROUGHNESS
SURFACE TEMPERATURE
SURFACE ORIENTATION
DEPOSIT ?
NO
NO ADVERSE SOILING EFFECT
YES
SURFACE PARTICLE ACCUMULATION
PARTICLE OPTICAL PROPERTIES
SURFACE OPTICAL PROPERTIES
SURFACE OPTICAL PROPERTIES ALTERED ?
NO
YES
OPTICAL EFFECT
CONTRAST
OBSERVER VISUAL ACUITY
OBSERVER COLOR PERCEPTION
OBSERVER DISTANCE FROM SOURCE
SENSED?
NO
YES
PERCEIVED ACCUMULATION
OBSERVER SOCIOECONOMIC STATUS
OBSERVER ATTITUDES (CULTURAL)
PURPOSE IN USE OF SURFACE
OFFENSIVE?
NO
YES
AESTHETIC OFFENSE
OBSERVER SOCIOECONOMIC STATUS
OBSERVER ATTITUDES
PURPOSE IN USE OF SURFACE
TAKE ACTION?
YES
NO
ECONOMIC EFFECT
(RESOURCES EXPENDED)
ECONOMIC EFFECT
(UTILITY LOSS)

Figure 4-1. Factors contributing to soiling effects of deposited particles.

Source: Bradow et al. (1985).

TABLE 4-3. SUMMARY OF LITERATURE ADDRESSING FACTORS CONTRIBUTING TO SOILING EFFECTS

Study	Pollutants Units (Term)	Pollutant Data Source	Index Term, Particle Surface Effect Addressed				Comments
			Physical	Sensory	Aesthetic	Economic	
O'Connor (1913)	Smoke, number of days/year visible smoke observed	U.S. Weather Bureau	NA[a]	NA	"It is safe to say that if it were possible to secure figures on 'the disagreeableness of it all' it would equal one-fourth of of the total of all the items of cost."	$/yr incremental cleaning costs associated with smoke	Related increased cleaning and associated costs to number of smoke days/year; Incremental cost of cleaning for Pittsburgh area estimated at $9.9 million/year
Michelson and Tourin (1966)	Air pollution, $\mu g/m^3$	Consumer Reports	NA	NA	--	$/yr incremental cleaning costs associated with particulates	Cleaning activity frequency and cost data gathered via survey; Task frequency related to particle concentration, and multiplied by task cost to estimate increment
Michelson and Tourin (1967)	Suspended particulates, $\mu g/m^3$	Not described; "some ...data obtained from Maryland Department of Health"	NA	NA	--	$/yr incremental cleaning costs associated with particulates	
Michelson and Tourin (1968)	Suspended particulates, $\mu g/m^3$	Connecticut State Department of Health	NA	NA	NA	$/yr incremental cleaning cost associated with particulates	

(continued on following page)

TABLE 4-3. (continued)

Study	Pollutants Units (Term)	Pollutant Data Source	Index Term, Particle Surface Effect Addressed				Comments
			Physical	Sensory	Aesthetic	Economic	
Carey (1959)	Atmospheric dust, tons/mi^2/mo	Calculated and from the Department of Scientific and Industrial Research (U.K.)	% area coverage	% area coverage noticeable by panel	% area coverage found offensive by panel	NA	% area of white horizontal surface covered by dust related to ambient particles by calculating deposition velocity; all particles contributing to effect assumed "fine" (<30 µm) and produced from fuel combustion Panel used to ascertain % of area coverage at which panel a) observed deposit and b) found deposit offensive; assumed rainfall every 10 days to accomplish outdoor cleansing; dusting every four days indoors
Narayanan and Lancaster (1973)	Dustfall, tons/mi^2/mo	Newcastle City (Australia) Department of Health	NA	NA	NA	$/yr incremental costs of household upkeep	Compared cleaning and maintenance costs for 2 areas differing in dustfall level
Esmen (1973)	Dustfall			NA	NA	NA	Proposed settled dust photometer for measuring soiling potential in terms of percent area per unit time
Hancock et al. (1976)	Dustfall, tons/mi^2/mo	Calculated from % effective area coverage rate	% effective area coverage	% effective area coverage detected by panel	% effective area coverage found offensive by panel	NA	Used panels to identify levels of dust coverage that determine thresholds of detection, discrimination, and identification

(continued on following page)

TABLE 4-3. (continued)

Study	Pollutants Units (Term)	Pollutant Data Source	Index Term, Particle Surface Effect Addressed				Comments
			Physical	Sensory	Aesthetic	Economic	
Booz, Allen and Hamilton, Inc. (1970)	Particulate matter, μg/m³	National Air Pollution Control Administration	NA	NA	Analysis included "attitudes toward cleanliness" as variables	Cleaning/maintenance task performance frequency	Found though frequency of cleaning in some cases related to suspended particulate level, expenditures for cleaning not related; did not consider value of homeowner labor
Beloin and Haynie (1975)	Total suspended particulate matter, μg/m³	Jefferson Co. (AL) Health Department	% change in reflectance (change in haze for glass)	NA	NA	NA	Building materials exposed for two years at Birmingham site; regression analysis showed time and particle concentration significant variables for reflectance change for white surfaces; color was additional significant factor for brick; poor correlations found for concrete, limestone, and window glass
Watson and Jaksch (1982)	Particulate matter, μg/m³	Booz, Allen and Hamilton	Used Beloin and Haynie and Esmen data.	NA	Used Booz, Allen, and Hamilton	$/year benefit (welfare gain), reduction of particulate level.	Used portions of Booz, Allen and Hamilton data to construct demand curve for household cleanliness; values derived from Esmen and Beloin and Haynie used for various supply curves; case worked for Philadelphia SMSA extrapolated to estimate national welfare benefit

[a] NA = not addressed in developing relationship between surface particle effect and/or ambient particle concentration.

Source: Bradow et al. (1985).

A necessary condition for soiling of surfaces is accumulation of a perceptible mass of contrasting particulate matter on a surface. This condition involves three physical properties of the deposited particles. Atmospheric particles can be characterized in terms of (1) deposition velocity, (2) concentration, and (3) color. The rate that particles accumulate on surfaces is related to deposition velocity and mass concentrations by the following expression.

$$\text{Accumulation Rate } (\mu g/m^2/sec) = \text{Deposition Velocity } (m/sec) \times \text{Concentration } (\mu g/m^3)$$

Deposition to the several types and orientations of surfaces typically encountered in the indoor environment has been studied very little, although there is some evidence (Sinclair and Psota-Kelty, 1985) that suggests that gravitational settling and Brownian diffusion are the dominant deposition processes. Since the wind speeds are negligible and the turbulence low indoors, extrapolation to the indoor condition for deposition velocities are not likely to be correct.

Generally the substances present in atmospheric aerosols have not been characterized by color, but probably the most intensely colored component of atmospheric particulate material is black elemental carbon or soot. Stevens et al. (1980) have found this material almost exclusively in the fine particle mass, accounting for more than 80 percent of fine particle mass in their analysis; other components, essentially colorless, were ammonium sulfates, nitrates, organic carbon, and a very small amount of lead. Wolff et al. (1983) found that elemental carbon concentration accounts for essentially all of the light absorption in filtered particles. Thus, the most highly colored substance present in atmospheric particles is present almost exclusively in combustion-produced fine particles.

Any model of soiling and its effect must interrelate physical, perceptual, and cost measures. As with any model, each measure must be on a common scale. Because scales available to physics and perception or behavior are often different, it is important to identify the appropriate scale type, and beyond that, to identify or at least consider the absolute threshold, the differential threshold, and context for perception. The significance of these factors for cost estimation can be illustrated. For example, if measures are extrapolated to the zero-zero origin to construct some linear model relating dirt deposited to dollar cost, costs would be underestimated. Most physical and psychological scales are not linearly related; in this case, the perceived dirt threshold begins at a value greater than zero physical dirt. Such a scaling procedure would produce an overly shallow slope for the prediction equation. A similar difficulty occurs if perceived dirt and physical amounts of dirt are assumed to be linearly correlated. These are almost surely related by a power function having an exponent less than one, in which case extrapolation by a model that extrapolates beyond data would produce prediction errors in the other direction (Bradow et al., 1985).

The "rationale for the secondary standards" discussed as part of the proposed NAAQS for particulate matter that appeared in EPA's Federal Register notice of March 20, 1984, provided a good summary of the state of knowledge regarding soiling effects on materials: "The available data base provides compelling evidence that elevated levels of particulate matter can produce adverse welfare effects, but provides little quantitative information on concentration-effects relationships. Physical damage and economic studies tend to show no obvious welfare effects "thresholds" for soiling... The available evidence suggests that the public makes a distinction between concentrations at which particulate pollution is noticeable and higher levels at which it is considered a nuisance." The notice goes on to state that no studies have established unique adverse particulate matter levels, however, and, on behalf of the EPA Administrator, "seeks the guidance of knowledgeable State and local air pollution officials with respect to levels at which the public appears to consider particulate matter a nuisance."

4.4 ODORS

The importance of the role of perception threshold identified in the preceding discussion of soiling as a welfare effect of indoor air is even more central to an evaluation of the effects on welfare of odors associated with the indoor environment. As noted in the discussion, perception threshold is not a property of the airborne particle. Similarly, the National Academy of Science (NAS) Committee on Odors from Stationary and Mobile Sources noted that the limit of detection, or odor threshold, is not a specific property of a substance, like its color or density. Instead, the threshold depends on the mode of presentation of the sample and on the sensitivity and even the expectation of the recipient. When such factors are carefully controlled, reasonably reproducible values can be obtained. A summary of olfactory thresholds identified for major air pollutants is provided in Table 4-4.

Odor intensity (the magnitude of the perceived sensation) can be described by an ordinal categorization, such as faint-moderate-strong. In more precise methods, numbering systems are used to estimate the magnitude of one intensity relative to another. One or more standard substances, in designated concentrations, may serve as references.

Attributes of odor other than detectability that are subject to measurement include odor intensity, character (quality), and hedonic tone (pleasantness-unpleasantness).

Odor character, or quality, is the property of the odor sensation that permits one to distinguish odors of different substances on the basis of prior exposure. Various systems of description have been proposed, and there have been some unsuccessful attempts to categorize all odors in terms of a small number of "primary" odor types.

The hedonic tone of an odor is the degree to which it is perceived as pleasant or unpleasant. Such perceptions differ widely from person to person, and adaptation is definitely a factor in tolerance. Furthermore, these judgments are strongly influenced by the previous associations that a person brings to the experience and by the emotional context in which the odor is perceived. Hedonic tone can be measured in terms of preference (dislike very much, like slightly, etc.), numbers, or pictorial references to facial expressions (smiling, frowning, etc.). Another approach parallels the estimation of intensity -- odors can be numerically rated in accordance with the degree to which they are more pleasant or unpleasant than other specified odors.

All these sensory methods require careful attention to the acquisition and preservation of a representative sample of the atmosphere or emission of interest and to the selection of appropriate human judges.

The chemical analysis of mixtures that contain many different chemical components requires the acquisition of a representative sample and the separation and identification of the components. To relate such information to the odor of the mixture, it is also necessary to determine which of the components are odorous and to assess their contribution to the intensity and character of the mixture. The analysis must be at least as sensitive as human olfaction.

TABLE 4-4. MAJOR ODOROUS AIR POLLUTANTS, OLFACTORY THRESHOLDS, AND RELATED DATA

Category	Class	Systemic Chemical Name	Formula	Mol. Wt.	Odor	Odor Threshold, ppm (by vol.)
Sulfur compound	Sulfur oxides	Sulfur dioxide	SO_2	64	Pungent	0.47
	Sulfides	Hydrogen sulfide	H_2S	34	Rotten eggs	0.0047-0.18
		Carbon disulfide	CS_2	76	Rotten	0.21-0.84
	Mercaptans	Methyl mercaptan	CH_3SH	48	Decayed cabbage	2×10^{-5}-0.041
		Ethyl mercaptan	C_2H_5SH	62	Decayed cabbage	3×10^{-5}-0.001
		Propyl mercaptan	C_3H_7SH	76	Unpleasant	0.0016-0.024
		Allyl mercaptan	$CH_2{=}CHCH_2SH$	74	Garlic	0.003-0.017
		Benzyl mercaptan	$C_6H_5CH_2SH$	124	Unpleasant	0.0026-0.04
	Thioethers	Dimethyl sulfide	$(CH_3)_2S$	62	Decayed cabbage	0.003
		Diethyl sulfide	$(C_2H_5)_2S$	90	Foul, garlic	0.0048
		Diallyl disulfide	$(CH_2{=}CHCH_2S)_2$	146	Garlic	1.1×10^{-4}-0.012
Nitrogen Compounds	Inorganic	Ammonia	NH_3	17	Pungent	0.47-54
	Aliphatic amines	Dimethylamine	$(CH_3)_2NH$	45	Fishy	0.047
		Trimethylamine	$(CH_3)_3N$	59	Fishy antebuccal	0.00021
	Aromatic nitro compounds	2,4,6-Trinitro-t-butyl-xylene (musk)	$C(C_4H_9)(CH_3)_2(NO_2)_3$	297	Musk	6×10^{-6}-0.005
	Heterocyclic amines	Pyridine	C_5H_5N	79	Empyreumatic	0.003-0.23
		Benzo[b]pyrrole (indole)	C_8H_7N	117	Fecal	0.05
		3-Methylindole (skatole)	C_9H_9N	131	Fecal	

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TABLE 4-4. (continued)

Category	Class	Systemic Chemical Name	Formula	Mol. Wt.	Odor	Odor Threshold, ppm (by vol.)
	Cyanides	Hydrogen cyanide	HCN	27	Bitter almonds	0.9
		Allylisocyanide	$CH_2=CHCH_2NC$	67	Sweet repulsive (nauseating)	0.18-1.6
		Allylisothiocyanate	$CH_2=CHCH_2SNC$	99	Mustard oil (nose and eye irritant)	0.008-0.42
Selenium Compounds	Selenides	Hydrogen selenide	H_2Se	81	Putrid	4×10^{-4}-0.0012
		Ethylselenomercaptan	C_2H_5SeH	109	Foul, fetid	4×10^{-4}-0.0012
		Diethyl selenide	$(C_2H_5)_2Se$	137	Putrid (nauseating)	0.011
Hydrocarbons, Alcohols, and Oxygenates	Aliphatic hydrocarbons	2-Butene (butylene)	$CH_3CH=CHCH_3$	56	Gas-house	24
		2-Methylpropene (isobutylene)	$CH_2=C(CH_3)_2$	56	Gas-house	20
	Phenol	Phenol	C_6H_5OH	94	Empyreumatic	0.047
	Aldehydes	Methanal (formaldehyde)	H_2CO	30	Pungent	1.0
		Ethanal (acetaldehyde)	CH_3CHO	44	Pungent	0.066-2.2
		Propenal (acrolein)	$CH_2=CHCHO$	56	Burning fat	0.021-1.8
		4-hydroxy-3-methoxy-	$C_8H_8O_3$	152	Sweet-aromatic	1.1×10^{-4}-2×10^{-7}
	Ketones	d-2-Keto-1,7,7,-trimethylnorcamphene (camphor)	$C_{10}H_{16}O$	152	Aromatic-earthy	1.3

(continued on following page)

TABLE 4-4. (continued)

Category	Class	Systemic Chemical Name	Formula	Mol. Wt.	Odor	Odor Threshold, ppm (by vol.)
	Organic acids	Butanoic acid (butyric acid)	$CH_3CH_2CH_2COOH$	88	Rancid, perspiration	0.001-2.2
		2-Methylbutanoic acid	$(CH_3)_2CHCH_2COOH$	102	Body odor	0.015
		Butanediene (diacetyl)	$(CH_3CO)_2$	86	Sweet butter	0.025
Halogen Compounds	Inorganic	Chlorine	Cl_2	71	Pungent	0.31
	Aliphatic halogens	Trichloroethylene	$CHCl=CCl_2$	131	Aromatic	0.21
		Triiodomethane (iodoform)	CHI_3	394	Antiseptic	5×10^{-3}
	Aromatic halogens	Benzyl chloride	$C_6H_5CH_2Cl$	126.5	Aromatic lacrimator	0.04-0.31
		Chlorohydroxybenzene (chlorophenol)	$C_6Cl_4(OH)Cl$	128.5	Medicinal	0.0036-0.03
Miscellaneous	Oxygen	Trioxygen (ozone)	O_3	48	Irritating	0.51

Source: National Research Council (1979).

Odors in combination tend to mask each other, so that the perceived intensity is weaker than sum of the individual odors' intensities (National Research Council, 1979). With regard to acceptable concentrations of odorous compounds indoors, in "the prevailing rule in buildings equipped with mechanical ventilation has been that the only acceptable odor is no odor at all" (National Research Council, 1979).

4.5 ECONOMIC EFFECTS

In evaluating the significance of welfare effects of air pollution, cost-benefit analysis has always been a major step in formulating regulatory decisions. Figure 4-2 depicts the relationship between pollutant emissions and economic damage. As shown, one may (1) proceed from ambient pollutant levels to economic damage estimates directly or (2) estimate damage based on physical damage functions. The latter route, called the damage function approach, has been the preferred method, although more recent studies have employed the first route. The estimation of willingness to pay is common to both choices.

Economic damage (benefit) that results from increased (decreased) pollutant concentrations can be estimated by willingness-to-pay approaches. All willingness-to-pay approaches try to estimate the aggregate monetary values that all affected individuals assign to the effects of a change in pollutant concentration. These approaches can be divided into three classes: damage function approaches, nonmarket approaches, and indirect market approaches. The first step of the damage function approach uses the relationship of pollutant exposure to physical damage. The second step links the physical damage to a dollar estimate of willingness to pay. Most economic damage estimates using this approach have not considered substitution possibilities for producers or consumers; however, proper consideration of these factors can yield good estimates of willingness to pay (via the damage function approach). Nonmarket approaches generally use surveys to ascertain the monetary values assigned to the effects. Indirect market approaches use information about the demand for marketed goods to estimate the willingness to pay for nonmarketed environmental attributes that are closely related to the marketed good (e.g., property value studies that estimate the willingness to pay for a change in the level of pollutant concentration through analyses of the changes in price of residential property). Each of these three approaches has different data requirements.

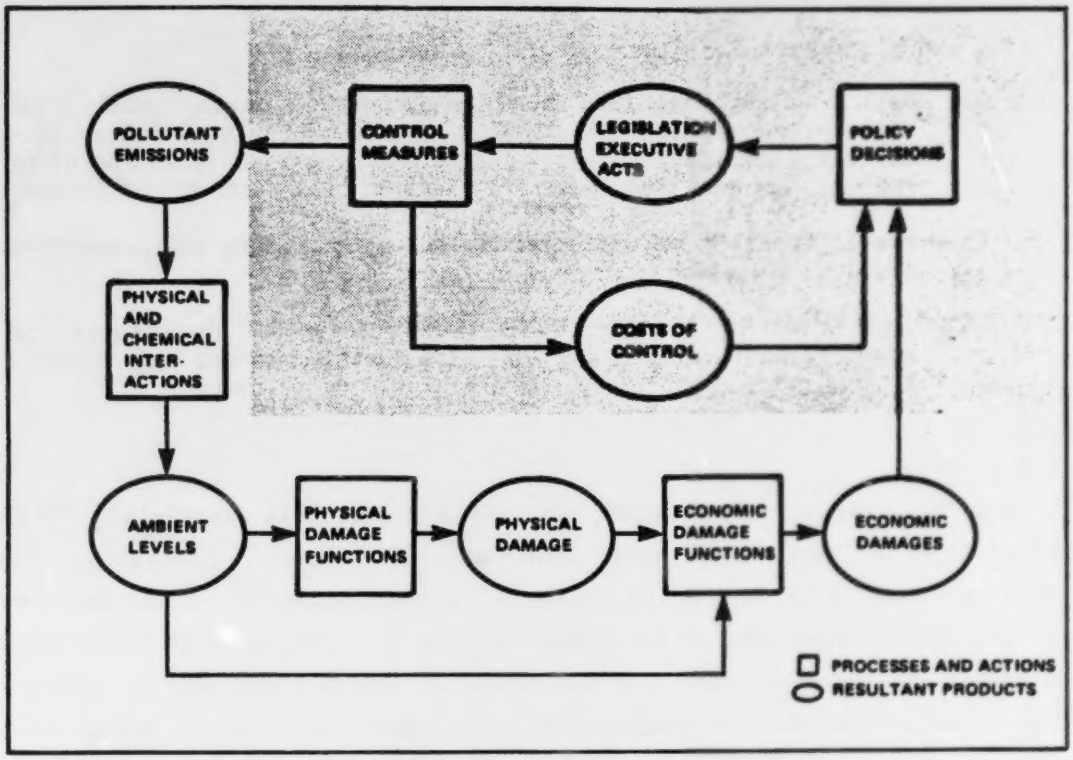
POLLUTANT EMISSIONS
CONTROL MEASURES
LEGISLATION EXECUTIVE ACTS
POLICY DECISIONS
PHYSICAL AND CHEMICAL INTER-ACTIONS
COSTS OF CONTROL
AMBIENT LEVELS
PHYSICAL DAMAGE FUNCTIONS
PHYSICAL DAMAGE
ECONOMIC DAMAGE FUNCTIONS
ECONOMIC DAMAGES
☐ PROCESSES AND ACTIONS
○ RESULTANT PRODUCTS

Figure 4-2. Relationship among emissions, air quality, damages and benefits, and policy decisions. Shaded area represents processes, actions, and resultant products outside the scope of this chapter.

Source: U.S. Environmental Protection Agency (1982).

Some of the considerations that must be taken into account when the analyses are used in the public policy decision making process include:

(1) selection of only the most important materials with significant damage as the subject of extensive damage cost analyses; those chosen for analysis must be both susceptible to high damage levels and represent major cost to society,

(2) when it is unlikely that a sufficiently significant reduction in pollution levels can be achieved, in cases where the smallest amount of damage is significant (such as in microelectronic components), elaborate analysis to develop refined data is unnecessary,

(3) the impact of pollution must be differentiated from the possibly even greater effects, of natural environmental factors,

(4) making assumptions that a given percentage of the total maintenance and replacement costs is attributable to pollution is a serious flaw; Many materials wear out or are replaced for other reasons before they are significantly damaged by pollution,

(5) the calculation of differences between avoidance and preventive costs is very difficult, and

(6) with works of art and historical monuments, the complex and aesthetic nature of their value to society renders cost/benefit studies of a quantitative nature highly questionable.

4.5.1 Radon

In the case of indoor radon, the economic impact is associated with health risks (real or perceived) rather than material damage. Since the government does not anticipate regulating levels of indoor radon, it is the decision of an individual homeowner as to whether or not to mitigate high radon levels. However, the economic impact on the value of the property may be beyond the homeowner's control. In areas where high indoor radon levels are prevalent, potential buyers as well as lending institutions may insist on radon testing. In some areas, lenders are currently requiring radon testing prior to closing on a home mortgage. Homes with high radon levels may decrease in value even if the levels have been reduced by remedial actions. Property values in an entire region may decrease if the region is perceived as having a potential radon problem. Once homeowners become aware of high radon levels, they may be legally liable for future damages if they sell the house without disclosing the problem. The potential economic effects of a radon scare on property values are tremendous.

5. SOURCE CATEGORIZATION

5.1 INTRODUCTION

The preceding chapters have summarized existing knowledge of indoor air quality. However, because there has been relatively little research attention given to the health risks of indoor air quality, the existing knowledge is fragmented, having been collected primarily for other purposes. For example, there is much known about NO_2 since it is regulated under the Clean Air Act, but little is known about NO_2 in complex mixtures with other existing pollutants from the same sources. Indeed, some of these indoor sources of NO_2 emissions, are not characterized adequately for the multitude of other emissions that comprise the mixture. Since much of the health information concerning indoor pollutants is derived from animal studies and is highly pollutant-specific, this information was summarized in Chapter 2 of this document by pollutant category. With the notable exceptions of ETS and non-ionizing radiation, most knowledge is pollutant-specific. In contrast, research needs are source-specific since the source causes the potential health risks that must be assessed, and the source or effects from the source are ultimately what must be mitigated. To choose a chemical-specific approach to indoor air quality research would, for the most part, be more expensive and time-consuming in nature, and would still leave unresolved the very major problem of how to assess the risks of complex mixtures from knowledge of a very few of the components of that mixture. Therefore, this chapter has been written with the goal of linking the chemical-specific approach of the Information Assessment document a source-specific approach for establishing research needs.

5.2 ENVIRONMENTAL TOBACCO SMOKE

Thousands of compounds (~3800) which have been identified in either mainstream (MS) or sidestream (SS) smoke are expected to be present in ETS. The concentration and phase distribution (particulate or vapor phase) will usually be altered in ETS as compared to either MS or SS as a result of dilution in indoor spaces. Undiluted SS contains a higher concentration of a number of toxic and carcinogenic substances than MS. Included are nitrosamines, aromatic amines, nicotine decomposition products, aldehydes, PAHs, CO, ammonia, NO_x, and many other nitrogen containing aromatics (e.g., pyridines, analines, and quinolines). ETS contains secondary reaction products not found in SS, and its composition may differ physically and chemically due to dilution, aging, and reactions with indoor surfaces such as walls, carpets and furnishings. Standard laboratory procedures have been established to characterize the properties of SS and MS. Research is still needed to standardize both the collection and evaluation of ETS so that the effects of ETS can be studied in laboratories and in human populations (National Research Council, 1986).

The changes in phase distribution of ETS constituents as the smoke is diluted and aged in the indoor environment are largely unknown. It is known, for example, that almost all of the nicotine shifts from the particulate phase in MS and fresh SS, to the vapor phase in ETS. Consequently, indoor air-cleaning devices designed to remove particles will not substantially alter the nicotine exposure, but may alter the concentrations of other toxic components. The particulate phase of ETS is dominated by small particles (<2.5 microns) referred to as RSP since they can be inhaled deeply into the lung. The exact amount of ETS retained in the respiratory tract and body of the nonsmoker is not known. Based on data from smokers, it is estimated that a nonsmoker exposed to ETS can retain 0.014 to 1.6 mg of RSP per day from ETS (Hiller, 1984). Studies of smokers have led to the hypothesis that hydrophobic vapor phase constituents of ETS (e.g., CO) are likely to enter the lung, while hydrophilic vapor phase constituents (e.g., acetaldehyde) are likely to be absorbed in the upper respiratory tract. Research is needed to determine the distribution of constituents in the particulate and vapor phases of aged ETS. Also, as discussed in chapter 3, the efficiency of air-cleaning systems in removing the constituents needs to be studied (National Research Council, 1986a).

Indoor radon decays to short-lived radon daughters, which may become bound to the RSP in ETS, thus affecting the human dosimetry of radon. Tobacco, however, contains some long-lived radon daughters. Research is needed to elucidate the possible interactions between ETS and radon daughters, especially as radon daughters can adhere to RSP and increase the potential hazard of ETS (National Research Council, 1986a).

Certain ETS constituents have been measured and used as surrogate indicators of ETS exposure in both personal and indoor air monitoring studies. Constituent compounds or classes which have been measured include RSP, CO, nicotine, NO_x, acrolein, nitroso-compounds, and benzo(a)pyrene. Unfortunately, many of these constituents are not good surrogates because they have a number of sources in the indoor environment other than ETS. Although nicotine and RSP have been particularly useful surrogates in the studies reported to date, no single measure has met the criteria for an ideal ETS surrogate. To facilitate health effects studies of ETS exposure, an ideal surrogate (marker or tracer) of exposure to ETS should be unique (or nearly unique) to tobacco smoke, should be present in sufficient quantities to measure at low ETS concentrations, and should be emitted in a reasonably constant ratio across brands and types of cigarettes to other tobacco smoke constituents of interest. Indoor air and personal exposure monitoring has been handicapped by the lack of a clear definition of the chemical and physical nature of ETS and the identification of the target constituents of ETS associated with the health and comfort effects. Reliable information needs to be obtained on the quantity, transport, and fate of such chemicals in ordinary indoor environments (National Research Council, 1986a).

The substantial emission rate of RSP from tobacco smoke has led to the conclusion that ETS is the dominant source of RSP indoors. For this reason a majority of field and human studies have used RSP as an indicator of exposure to ETS. In personal monitoring studies, total RSP has been found to be substantially elevated for individuals who report being exposed to ETS as compared to those who report no such exposure. Air monitoring and modeling studies both indicate that RSP levels will be clearly elevated over background levels in indoor spaces when even low smoking rates occur. In future indoor air research it is recommended that the importance of variation in the input parameters such as room size, temperature, humidity, air exchange rate, and numbers of cigarettes smoked should be noted when interpreting the data on constituents of ETS obtained from personal monitors and indoor space monitors (National Research Council, 1986a).

Exposures to ETS have been assessed by questionnaires, air monitoring, estimation through modeling of concentrations, and biological markers. The simplest and yet the least precise and reliable method of exposure assessment has been the use of questionnaires. Such questionnaires have been the basis for classifying individuals into broad categories of exposure, however there are serious difficulties in developing uniform questions that elicit unambiguous and correct replies, and even more difficult problems in using these replies to make quantitative estimates of exposure. Questionnaires are particularly difficult to use to estimate an integrated exposure over many years, yet this is the primary method which has been used to approximate such long-term exposures. The National Research Council (1986a) recommends that future epidemiologic studies should incorporate into their design several of these exposure assessment methods in order to assess exposure to ETS more accurately and to allow estimation of dose. To estimate integrated exposure to ETS, future studies need to estimate a long-term ETS exposure history, including which fraction of the day is spent in the presence of ETS and at what ages these exposures occurred. The data from such a history should be entered into a specific time-place model, from which cumulative exposure can be estimated.

Personal exposure and dosimetry of ETS is dependent upon so many factors that the optimal assessment should use biological markers that accurately indicate uptake and/or dose in physiological fluids, tissues, or cells. Several chemicals found in body fluids of active smokers have been evaluated as biological markers of exposure to ETS, and the National Research Council (1986a) recommends that other markers be investigated as described below. The criteria for acceptable biological markers are similar to those for measuring ETS in the external environment.

Nicotine and its metabolite, cotinine, measured in saliva, blood, or urine, have been the most useful biological markers of recent ETS exposure, because they are derived virtually exclusively from tobacco products. Urinary cotinine levels have been shown to increase in nonsmokers with increasing number of smokers in the home for all age groups. Currently there is difficulty in interpreting the relative cotinine levels in nonsmokers compared to smokers because of the reported slower clearance of cotinine in nonsmokers and the lack of good uptake and clearance data for nonsmokers of different ages, sex, and genetic backgrounds. The National Research Council (1986a) recommends that absorption, metabolism, and excretion of ETS constituents, including nicotine or cotinine, need to be carefully studied in order to evaluate whether there are differences between smokers and nonsmokers in these factors. Further epidemiologic studies using biological markers are needed to quantify exposure-dose relationships in nonsmokers.

Several other potential biological markers that have been evaluated as indicators of ETS exposure are thiocyanate, COHb, and exhaled CO. These compounds have not been found in sufficient quantities in body fluids at moderate or low levels of ETS exposure to be generally useful. Since there are several other sources of CO in the environment that equal or exceed the contribution made by ETS, this marker is even less useful. Although the use of nicotine and cotinine measurements in urine, and possibly saliva, are recommended as the best methods now available for quantifying human exposure to ETS, these are not ideal markers for all constituents of ETS. In ETS polluted environments, nicotine is currently thought to be present in the vapor phase as a free base, thus its uptake by the passive smoker may not be representative of the uptake of acidic and neutral smoke components from the vapor phase nor of any component in the particulate phase. Other suggested biological markers of exposure include N-nitrosoproline, nitrosothioproline, and some of the aromatic amines that are present in high concentrations in SS, as well as 3-vinylpyridine, solanesol, and other tobacco specific constituents. Thus, the National Research Council recommends that future studies should be concerned with developing techniques to measure the uptake by nonsmokers of various other types of tobacco-specific ETS components which would be representative of the particulate organic phase of ETS and the volatile acidic and neutral phases.

Another type of biological marker of ETS exposure is genotoxicity of the urine. The National Research Council (1986a) concluded that on the basis of presently available data, it is likely that the exposure of nonsmokers to heavy ETS increases the potential for mutagenic activity of their urine which is elevated above that which is observed in the same nonsmokers before, and long after, ETS exposure. The evaluation of mutagenicity in the urine of nonsmokers as a result of ETS exposure must consider the possibility of confounding factors such as dietary constituents, occupational exposures, and other environmental exposure factors which may render the findings of elevated mutagenicity as nonspecific. Research is needed to clarify the appropriate methods for estimating mutagenicity (particularly ETS-specific urinary mutagens) and to isolate and identify the active agents in the body fluids of ETS-exposed nonsmokers (National Research Council, 1986a).

Highly sensitive methods are now becoming available for determining protein or DNA-adducts of environmental carcinogens and toxic agents in circulating blood and tissues. Several constituents which occur in ETS, e.g., benzo(a)pyrene and 4-aminobiphenyl, have been reported as hemoglobin or DNA adducts, however these chemicals are not specific or unique to ETS. The development and validation of methods to detect ETS-specific adducts would provide an ideal marker of human exposure and in some cases (e.g., DNA-adducts), dose of ETS. The NRC concludes that validation and quantitative determination of the uptake of tobacco smoke carcinogens is urgently needed. Studies are needed to develop and apply highly sensitive methods (e.g., immunoassays or postlabelling) for measuring DNA and protein adducts of tobacco-specific chemicals.

Evaluations recently completed by the NAS (National Research Council, 1986a) and the Surgeon General of the U.S. (1986) both concluded that considering the evidence as a whole, exposure to ETS increases the incidence of lung cancer in nonsmokers. All of the studies from various countries, including the United States, consistently show a 30 percent increased risk (within 95 percent confidence intervals) for nonsmoking spouses of smokers. These estimates are almost all derived from the comparison of persons identified as exposed or unexposed on the basis of their spouses smoking habits determined by questionnaires. Both reports concluded that better data are needed on the extent and variability of ETS exposure and the associated dosimetry in order to accurately estimate the number of deaths or magnitude of risk in the U.S. population. Because more than 135,000 deaths from lung cancer are expected in the U.S. in 1986 alone, the Surgeon General's report (1986) concluded that the number of lung cancer deaths attributable to ETS (passive smoking) is substantial and represents a problem of sufficient magnitude to warrant substantial public health concern. Both groups concur that laboratory studies are needed to determine the concentrations of carcinogenic constituents of ETS present in typical daily environments (e.g., indoor air). The use of biological markers in epidemiologic studies is recommended to quantify more precisely the dose-response relationships between ETS exposure and lung cancer occurrence.

Laboratory studies have contributed to a better understanding of the factors and mechanisms involved in the induction of cancer by tobacco smoke. There have been many bioassays conducted on MS (and often on cigarette smoke condensate, CSC) including genotoxicity assays, carcinogenicity assays, and other bioassays related to either tumor initiation or tumor promotion. Skin tumorigenesis assays, although using a route of exposure different from the human respiratory tract, have been especially useful in evaluating the carcinogenicity of different tobacco constituents. Similar studies with skin painting have not been done with ETS and would be of value for assessing the differential toxicity (e.g., tumor-initiating and tumor-promoting activity) of ETS and MS (National Research Council, 1986a). Only one study has examined the carcinogenic potential of the condensate of sidestream cigarette smoke (Wynder and Hoffmann, 1967). The results of this study indicated that SS was more tumorigenic than MS.

ETS exposure involves proportionately more exposure to gas-phase than to particulate phase constituents when compared to MS exposure. There have not been laboratory studies of the effects of exposure to ETS *in vivo*, and very few studies have compared MS, SS, and ETS in short-term *in vitro* assays. Both reports recommend that additional laboratory *in vivo* and *in vitro* studies be conducted to compare the mutagenicity and carcinogenicity of MS and SS, and that further studies be initiated to evaluate the various constituents of ETS (e.g., gas phase versus particles, specific fractions) of ETS.

There is no consistent evidence at this time of any increased risk of ETS exposure for cancers other than lung cancer. Future epidemiologic studies need to examine the potential risk for nonsmokers exposed to ETS for cancers such as brain, hematopoietic, nasal sinus, or other cancers consistently related to active smoking, and cancers of all sites. The NRC recommends that lymphohematopoietic neoplasms should be studied in relation to ETS exposure, particularly as a result of childhood exposure and any potential interrelationship or interaction with radon exposure.

Acute irritating effects of ETS, especially of the eyes, but also of the nose and throat, are the most commonly reported effects. These effects have been documented by studies in which individuals are asked questions regarding odor or irritation or by measuring eye blink rates. The National Academy of Sciences (National Research Council, 1986a) recommends that objective physiological or biochemical indices should be sought to validate reports of noxious reactions and chronic irritation associated with ETS. A number of studies using standard dermatological tests have shown that tobacco smoke contains immunogens, and individuals report allergic-like responses to exposure to ETS. Further research is needed, however, to evaluate the medical importance of positive skin tests to ETS extracts and to relate immune response on skin tests to subjective complaints about the noxious, irritating properties of ETS (National Research Council, 1986a). Although there is some evidence that it is the gaseous components of ETS that are the most irritating and objectionable, more research is needed to determine the specific constituents that are the irritants and/or allergens in ETS.

Respiratory effects, both acute and chronic, have been shown to be related to exposure to ETS in children, particularly those exposed to ETS during the first two years of life. These effects include pulmonary symptoms (e.g., wheezing, coughing, and sputum production) as well as respiratory infections (e.g., manifested as bronchitis and pneumonia). Children whose parents smoke have measurable effects on lung function; however, it is not clear whether these children exposed to ETS will be at increased risk for the development of chronic obstructive lung disease. Both the Surgeon General (1986) and the National Research Council (1986a) recommend that ETS be eliminated from the environments of small children. Because much of the evidence for adverse respiratory effects of ETS on infants and young children is derived from studies of children whose parents smoke, it is difficult to conclusively demonstrate whether these effects result from indoor air exposure to ETS, because the children were often exposed _in utero_ as well. More research is needed to clarify the respiratory effects of ETS exposure postnatally. Studies are also needed to distinguish the effects of ETS on asthmatic and hyperresponsive individuals. The National Research Council (1986a) recommends that a combination of animal toxicological studies and human studies be used to address these unresolved issues.

Although cardiovascular disease is associated with active smoking, both the National Academy of Sciences and Surgeon General conclude that further studies are needed to establish a causal relationship between either acute or long-term ETS exposure and such disease. The connection between cardiovascular disease indicators such as cardiac function, blood pressure, and angina in nonsmokers and ETS exposure must be made before an effect of ETS on the etiology of cardiovascular disease can be established.

Several other health effects of ETS have been studied in children, particularly effects on growth and development. In most of these studies it has not been possible to differentiate the _in utero_ exposure from the indoor air ETS exposure during infancy and childhood. Effects on growth and development need to be investigated before conclusions can be drawn about such effects from ETS exposure. A number of studies consistently report that chronic middle ear infections are more common in children whose parents smoke than in children of nonsmoking parents. The association between chronic middle ear infections and ETS exposure has not yet been causally established, however.

5.3 BIOLOGICAL CONTAMINANTS

Biological contaminants such as viruses, bacteria, fungi, and protozoans can originate as infectious agents shed by humans and animals. These same groups of organisms, as well as insects, arachnids, and acarids, can originate outdoors, but colonize indoor spaces under proper conditions of nutrition, moisture, and temperature. Pollens, too, can enter indoor spaces through cracks. Data concerning sources and health effects, as well as mitigation efforts are detailed in Chapter 2.

At present, significant information about levels of pollution by biological contaminants is not available. There is no baseline data of what constitutes "normal" levels of flora, such as bacteria and fungi. Monitoring instruments and techniques are primitive. Techniques for monitoring biologic contaminants, especially viable agents, have not been standardized, so that comparison of monitored levels by different researchers is not possible. Seasonal and geographic patterns and variations in levels of organisms are not known. Many fungi produce mycotoxins with very high toxic potentials that in many cases are also carcinogenic. The conditions under which these toxins are produced and the levels and kinds of toxins found indoors are generally not known, but some neurotoxic symptoms from some mycotoxins mimic those of "sick building syndrome". Some bacteria produce endotoxins, for which the health effects in indoor environments are not known.

The virulence of viable organisms varies tremendously, so that a single organism can be infective for some diseases, while others require many thousands of organisms per liter to be infective. Again, baseline data for virulence of viable contaminants is not known.

Almost all biologic contaminants have allergenic potential. This includes dander and excreta from humans and animals, as well as body parts and secretions from insects, arachnids, and acarids. Pollens and fungal spores also have allergenic potential. The numbers and sensitivities of people sensitized to such agents is not well defined.

5.4 PERSONAL ACTIVITIES

Pollutants generated from personal activities include NO_2, particles, biological substances, and gas-phase organic compounds. Further descriptions of these pollutants can be found in Chapter 2 of this document. Indoor NO_2 levels are invariably high in the presence of smokers and cooking with gas relative to outdoor values. Currently, integrated exposure to NO_2 can be measured by two passive devices: the Palmes tube and the Yanagisawa badge, and by two types of commercial personal monitors. Animal toxicology studies support the belief that chronic exposure to NO_2 may result in chronic lung disease. Moreover, the concentration of NO_2 has more of an effect on overall health than the duration of exposure.

Particles and biological substances are generated by personal activities such as vacuum cleaning and sweeping where the particles are reentrained from floors and rugs. House dust, especially that collected in carpet fibers, serves as an important sink for air pollutants, as well as soil particles brought into the home by foot traffic. Extensive particulate sampling has been conducted in the ambient environment, but generally these methods and samplers are not well suited to indoor monitoring. A particulate exposure monitor currently being developed by EPA maintains the integrity of organic chemicals in house dust. There is insufficient knowledge of personal activity patterns indoors to quantify the relationship between particulate matter exposure and dose. Validated, standardized methods for biological aerosols are needed for indoor use.

Gas-phase organic compounds originate as products from personal use activities involving hair spray, paint solvents, and cleaning fluids. The TEAM studies conducted by the U.S. EPA utilized personal monitors with Tenax® sampling cartridges and miniature air pumps to collect VOCs. Generally it is thought that the levels of individual VOCs are well below threshold limit values considered to be harmful for any compound.

The contribution of pollutants generated by personal activities to total indoor exposure is largely unknown. Additional research is needed on determining personal activity patterns indoors, with subsequent measurement of spatial and temporal gradients of pollutants associated with personal activities. Moreover, existing measurement methodology may not be adequate to meet the special demands of personal exposure monitoring. The development and validation of quiet, unobtrusive devices is needed to accurately assess exposure to many indoor pollutants.

5.5 BUILDING SYSTEMS

In spite of the recognized importance of the building system, there is little information on building system performance. Most IAQ studies are either limited to studies of sources or to individual building studies. These building studies clearly demonstrate the difficulties of diagnosing IAQ problems. These studies also demonstrate that many IAQ problems are mixture problems. That is, the problems are caused by a mixture of pollutants and by the interactions of these mixtures with the building system.

The role the building system plays in determining IAQ must be better defined. The research necessary to do this falls into six categories. These are:

- ventilation and ventilation effectiveness
- component performance
- building system source and sink effects
- effects on sources and sinks
- effects of building systems on health effects
- integration of data from diverse sources into understanding of whole system.

Each of these needs is discussed briefly below. Ventilation is a major theme in all IAQ work. Increased ventilation is offered as a mitigation method for nearly all problems, yet there is no clear understanding of how to make maximum use of ventilation to improve IAQ. In many cases, gross building ventilation is adequate (i.e., standards are met), but IAQ is poor due to ineffective utilization of the ventilation. Work is needed to develop measurement methods to define ventilation and ventilation effectiveness. Research is also needed to improve ventilation effectiveness.

A building system is made up of numerous components. The performance of many of these components (e.g., air cleaners) is not well understood--especially under conditions of interest in IAQ. Work to quantify the performance of major components of the building system is essential.

The building system can act as a source or a sink for many pollutants, yet there are no good data describing the role of the building system as a source or a sink. Such data are essential to a clear understanding of IAQ. A coordinated research effort to develop these data is necessary. This research program must be coordinated with other source type research to avoid overlap. Research on various sources and sinks must be conducted to ensure that these effects are quantified.

The building environment can also have a major impact on the health effects from various pollutants. However, there is insufficient information on the exact nature of these impacts. Thus, research is needed to define the effects of building environment on health effects of various pollutants.

A full understanding of IAQ requires that the building system and the various separate sources and sinks be treated as a whole. This integration of the various pieces of data into a complete understanding of IAQ requires research to develop models and simulators of buildings. The building simulators should include methods to predict health impacts.

5.6 MATERIALS AND FURNISHINGS

Formaldehyde is the only major organic vapor for which data are adequate to characterize sources and associated emission rates from building materials and furnishings. Major uncertainties exist for other organics and their sources. One area which has received little attention is that of transportation. Since the public spends significant time in motor vehicles and airplanes, data on organic vapor sources in these environments is needed. These uncertainties lead to the following research needs for source characterization. Testing protocols are needed for determining emission rates from vehicle sources via environmental test chambers, including consideration of temperature, relative humidity, air exchange, air exchange, product loading, chamber vapor concentration, and chamber wall effects.

Emission factors are needed for a wide variety of building materials, furnishings, household chemicals, and consumer products; such emission factors should include consideration of environmental variables and should be applicable for total organics and major organic species. The time variability of emissions should also be examined, and materials and products used in transportation microenvironments should be included.

Source models should be developed which relate emission factors to indoor concentrations; such models would be inputs to more general IAQ models discussed elsewhere. Model testing and validation would be necessary in test house and field studies.

Material and product rankings should be developed based on their emission characteristics (e.g., emission rates and compounds emitted); these rankings could be coupled with health effects information to develop rankings based on health hazards.

While the effects of ventilation on reducing indoor concentrations of organic vapors by dilution and flushing are fairly well documented, the effects of changing air exchange rates on source emission rates is unclear for many sources and compounds. Emission rates for some compounds can actually increase with increased ventilation. The effectiveness of building "bake out" to achieve emission reductions has yet to be determined. Air cleaning devices employing adsorption or catalytic oxidation are well understood for single compounds at concentrations exceeding 100 to 1000 ppm, but performance data are needed for such devices at lower concentrations (e.g., 1 to 100 ppb, or roughly 5 to 500 $\mu g/m^3$), and for mixtures of organic vapors. The following research needs for organic vapor control are suggested.

The influence of material and product age on emission rates should be determined; such information would be developed via long-term testing in environmental test chambers. The influence of ventilation practices on emission rates for individual compounds needs to be determined. The effectiveness of building "bake out" via environmental chamber testing at elevated temperatures and air exchange rates should be evaluated; test house and field studies are necessary for validation. Finally, the effectiveness of air cleaning devices, including absorbers and catalytic oxidizers, should be determined.

5.7 COMBUSTION APPLIANCES

Combustion appliances such as gas stoves, heaters, and water heaters, kerosene heaters, woodburning stoves, furnaces, and fireplaces are in common use across the United States. All these sources involve combustion at some stage and produce both indoor and outdoor pollution, the specific magnitude being dependent on the degree of venting to the outside and/or the degree of leakage indoors.

Indoor combustion of all organic material is incomplete, resulting in the production of NO_x, CO, CO_2, and a very broad class of substances called "products of incomplete combustion" (PICs). For some organic fuels, combustion emissions are enriched in SO_2 (e.g., kerosene or high-sulfur gas, oil, or coal).

It is not possible to assess adequately the health risks associated with exposure to combustion emissions at the present time for several major reasons. Exposure assessment to PICs is not yet possible. A significant number of studies have monitored emissions from combustion appliances, especially gas stoves and kerosene heaters, but the monitoring has focused on the criteria pollutants within the emissions (i.e., NO_2, CO, SO_2, PM). Even so, there is significant uncertainty of what the exposure patterns are on an hourly (or other relatively short-term) basis. Some research has focused on PICs, since several are carcinogenic, but this work has been relatively small in scope and methods are not adequate presently to characterize the majority of PICs. Dosimetry provides a linkage between exposure and hazard assessment, (i.e., the relationship between exposure level and delivered dose which produces the health effects), but it is insufficiently studied to be of impact in risk assessment. Knowledge of dosimetry is reasonably advanced and/or part of an ongoing effort for NO_2 and for insoluble particles. However, insufficient research attention is being focused on dosimetry of PICs, including the organics absorbed onto particles. Hazard assessment is likewise deficient. There is substantial knowledge of the health effects of individual gaseous pollutants such as NO_2, CO, and SO_2, but even if there were adequate information on NO_2 indoor exposure patterns, for example, the risk of exposure to the source would be unknown due to toxicological interactions within complex mixtures. Current health information on PM is specific to the outdoor environment. Of the known PICs, many have been studied for mutagenicity and a few for carcinogenicity, but many have not been studied at all and still more PICs have not even been identified. A more specific discussion of these issues as related to individual combustion appliances follows.

Of gas stoves and heaters, gas stoves have been the most frequently studied. The research has focused on NO_2, since relatively high levels of NO_2 (compared to outdoors) are emitted by combustion appliances, and NO_2 is a NAAQS pollutant. Numerous monitoring and epidemiological studies of gas stoves have been completed (see Section 2.2.4.4). The results of the studies are controversial, primarily due to the conflicting results which might be traceable to the variation in methods and study approaches applied. On balance, these studies do indicate that there is a health risk from cooking with unvented gas stoves, but this health risk is inadequately characterized from both exposure assessment and hazard assessment perspectives.

Gas heaters have long posed an "accidental" health risk since high levels of CO can be produced which have resulted in significant illness and even death. These "accidental" risks have been known and mitigation procedures established. However, risks from routine use are not adequately characterized.

Kerosene heaters have been more recently studied to determine emissions and the mutagenic (and therefore possible carcinogenic) risks. Results indicate that relatively high levels of SO_2 are emitted, causing concern that the pulmonary function of asthmatics might be affected. Organic fractions of the emissions of a few of the kerosene heaters studied are mutagenic in *in vitro* bioassays. The information available suggests the possibility that some carcinogens such as nitro-PAHs and HCHO may be emitted under some combustion conditions. There are several types of kerosene heaters: convective, radiant, radiant/convective, and wickless, which are also categorized as blue flame and white flame heaters. Further complicating risk assessment is the variation in fuel type, wick height, length of operation, and design of the unit. Because of these variables, there is a wide variety of exposure situations, none of which have been adequately characterized. There is also a wide variety of emitted pollutants, of which only a few have had health effects characterized. Those that are known are sufficient to cause concern over health risks. The major uncertainties for both carcinogenic and noncarcinogenic risks need to be reduced to enable hazard assessment. Research to address these issues can focus on specific alleviation or mitigation recommendations to be applied either by engineers or homeowners. For example, if one fuel type is particularly hazardous, a less toxic fuel might be used.

Wood burning has become increasingly common. For example, in the Pacific Northwest, up to 50 percent of homes use wood fuel to meet some of their heating needs. Woodburning appliances are vented to the outside, but there is an indoor pollution component because even "airtight" wood heaters are a statistically significant source of indoor PM and PAHs, several of which are carcinogens or have carcinogenic potential. NO_x and CO, each having noncarcinogenic health risks, are also emitted. Non-airtight combustion units (especially fireplaces) contribute more to indoor pollution than the airtight units. As is the case for other classes of combustion units, there is sufficient evidence to be concerned about the health risks, especially carcinogenic risks, for wood stoves and fireplaces, but concern alone does not enable risk assessment and does not advise manufacturers or the public about design and/or use of units to mitigate that risk.

The increasing public use of combustion appliances is caused principally by economic considerations, since use of electrical units with similar functions is more expensive. There is a counterpart economic decision, namely the impact of health risks, that needs to be incorporated in decision-making by the public. It is also important to advise manufacturers about redesign options that might reduce health risks. Generally, there is no doubt that there is a health risk, the doubt resides in the characterization and degree of health risk. Such uncertainties must be clarified to permit informed decision-making by the public. To these ends, a research program has been proposed which begins with a preliminary comparative assessment of health risks from major types of combustion appliances. This approach produces information for preliminary decision-making by the public and, equally important, provides guidance as to whether further research is necessary and if so what its direction should be.

5.8 OUTDOOR SOURCES

5.8.1 Radon

It is generally agreed that the principal source for high indoor radon levels is the soil under the structure. The radon enters the building as part of the soil gas which is driven by the pressure differential between the inside of the building and the soil. In a few locations, the radon level may be increased by the release of dissolved radon brought into the home in the potable water supply. However, in the case of private water supplies (which is the only category with a significant problem), less than three percent of the average background indoor radon comes from the water. If potable water is likely to be a significant source of radon, a number of mitigative techniques have been proposed (Lawrence Berkeley Laboratory, 1982; U.S. Environmental Protection Agency, 1984b). These include: 1) alternate water supplies, 2) reconstruction of wells, 3) radon removal treatments, 4) aeration facilities, and 5) the use of granular activated carbon beds.

In a few special instances, significant radon contributions come from building materials. Avoidance of building materials made from uranium or phosphate mining wastes is recommended.

In order to develop procedures for diagnosing radon entry points and factors affecting entry, both before and after radon reduction techniques have been employed, it is necessary to carry out a comprehensive measurement program in a variety of contaminated houses. Without careful measurements to characterize the mechanisms which influence radon entry, the mitigation process will remain an art form practiced only by a select group of building contractors rather than the science it could become. To illustrate that house mitigation is not yet a science, we need only point out that common mitigation techniques which work well in some situations often fail in other apparently similar situations, and the reasons for failure are not well understood. With careful measurements it should be possible to understand why one application is successful while another is not. In fact, with a little knowledge of the driving mechanisms, the differences might prove to be predictable.

Several advances in controlling indoor radon concentrations have been made in recent years. The EPA has done a significant amount of work on radon mitigation in existing homes and a guidance document for homeowners has been issued (U.S. Environmental Protection Agency, 1986d). Some of the mitigation methods under study include: 1) Natural and forced air ventilation, 2) avoidance of structure depressurization, 3) sealing of potential soil gas entry routes, 4) forced air ventilation with heat recovery, 5) drain-tile soil ventilation, 6) hollow-block basement wall soil ventilation, and 7) sub-slab ventilation. Air-cleaning systems that use air filtration or electrostatic precipitation have also been studied. However, because their effect on the actual radon dose to the lung is unclear, air-cleaning techniques are less desirable radon control methods. Source reduction or increased ventilation are more favored means of reducing indoor radon concentrations. The majority of the mitigation techniques deal with reducing soil gas entry because this is known to be the most significant radon source.

Since soil gases are the major sources of indoor radon, there is a compelling need to understand the mechanisms by which radon collects and migrates in specific soils and geologic formations as well as the factors which are rate controlling. The ability to predict the emanation characteristics from different types of soils would be quite valuable in locating houses with very high radon levels as well as locating potential new building sites. Unfortunately, this predictive capability does not yet exist, but it should have priority on the list of research needs.

In many respects, designing a "radon-resistant" home should be much easier than retrofitting an existing one against radon entry from soil sources. Building a new house on low radon-emanating soils would be the obvious first choice, however predicting the soil-emanation characteristics is not so easily accomplished and significantly more work needs to be done toward this goal (American ATCON, 1986). The knowledge which the EPA has gained by studying existing houses is now being applied to the design of new houses. The choice of construction methods for new homes in radon-prone areas should be given careful consideration. For instance, hollow concrete blocks should not be used for basement walls or in the foundation, and all cracks and openings should be sealed. Gravel drainage beds with perforated drainage pipes should be installed on all four sides of the house and all drains should be sealed with a water trap. It is also advisable to put good aggregate under any concrete slab (American ATCON, 1986). An important aspect of EPA's continuing research is identifying the most cost-effective radon-reduction methods for particular structures and soil characteristics and transferring the technology to both the private and the public sectors.

The major aspects of current research needs in the reduction of indoor radon levels are summarized in the following statements. Development of procedures for diagnosing radon entry points as well as pertinent factors affecting its entry, both before and after the installation of reduction techniques, are urgently needed. Development and demonstration of cost-effective methods for reducing radon concentrations in existing houses are needed, as are development and demonstration of cost-effective techniques to prevent high radon levels in new houses. And finally, the transfer of technical information on the design, installation, operation, performance, and cost of radon reduction methods and equipment to the Federal, State, and Local governments, as well as to the private and public sectors, is essential.

5.8.2 Pesticides

While present data on the occurrence and sources of indoor pesticides is sparse, the ongoing NOPES investigation will provide substantial data. Research is needed to determine emission rate characteristics, including: 1) development of testing protocols for determining pesticide emission rates via environmental test chambers, 2) development of pesticide emission factors, and 3) development of pesticide source models, including testing and validation.

Only limited data are available on the effects of ventilation on indoor pesticide concentrations. No data were found on the use of air cleaners to control pesticide levels. Limited data are available on the influence of application practices on indoor pesticide exposure. These data gaps lead to the following research needs:

- determining the influence of ventilation practices on pesticide emissions to the indoor environment,
- evaluating the impact of pesticide application practices on the indoor residuals,
- determining the applicability and effectiveness of air cleaners for removing or destroying indoor pesticide vapors, and
- evaluating radon reduction techniques for the control of indoor chlordane levels (Chlordane, from soil treatment for termites, has the same entry routes as soil radon; thus, techniques presently being demonstrated for radon mitigation should be effective for chlordane control).

5.9 CONCLUSION

The goal of reducing risk by reducing total exposure to air pollutants can be approached by source control and mitigation. Table 5-1 indicates some of the relationships between chemical-specific pollutants and their source-specific origins. The Research Needs Document, which follows, addresses indoor air pollution abatement from a source specific approach, as well as a more generic approach which encompasses several source categories.

TABLE 5-1. RELATIONSHIP BETWEEN POLLUTANT AND SOURCE CATEGORIES

Pollutants	Passive Smoking	Combustion Appliances Vented and Unvented				Materials and Furnishings				Sources of Biological Contamination	Personal Activities					Outdoor Environment
		A1	A2	A3	A4	B1	B2	B3	B4		C1	C2	C3	C4	C5	
Tobacco Smoke Components	X															
Radon	0					X				X			0	0		X
Particulate Matter	X			X						X	X	X	0	X	X	X
TSP																
RSP																
PM_{10}																
NO_x	X	X	X	X	X							X			X	X
SO_6																
SO_4																
CO	X	X	X	X	X							X	0		0	X
Oxidants	0	X	X	X	X							X	0	X	X	X
Pb	0											X	0		0	X
CO_2	X	X	X	X	X				X			X	0			X
Metals	X											X	0	0	X	X
Fibers	0					X		X			X	X	0	X	X	X
Asbestos																
Synthetics																
Biological	0									X						
Bacterial																
Fungi																
Plant Spores																
Insects																
Pesticides	0												X			X
Organic Compounds (Non-ETS)	0	X	X		X	X	X	X	X		X	X	0	X	X	X
PAHs																
Aldehydes																
Alcohols																
VOCs																
CFCs																
Hydrocarbons																

A1 - Gas Heaters
A2 - Kerosene Heaters
A3 - Wood/Coal Stoves and Fireplaces
A4 - Gas Cooking Stoves

X = Direct Source

B1 - Building Materials
B2 - Household Chemicals
B3 - Furnishings
B4 - Stored Materials

C1 - Personal/Office Products
C2 - Transportation
C3 - Athletics
C4 - Maintenance (Cleaning, Painting)
C5 - Misc. (e.g., Photocopying)

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ATTACHMENT 1. RESPONSE TO ISSUES FROM HARVARD WORKSHOP, JANUARY 1987

At the request of the U.S. EPA's Office of Research and Development, Harvard University organized a workshop to review ORD's Preliminary Assessment for the Indoor Air Program. A group of 20 of the world's leading researchers on indoor air pollution, Harvard staff and EPA personnel participated in this review. The format of the workshop was that the attendees were divided into small groups which focused discussion on the information compiled on specific pollutants. Each group leader then summarized the work of the group in a series of viewgraphs, which were then presented for discussion by the assembled participants at two plenary sessions.

A number of general recommendations were made concerning the document as a whole, as well as suggestions concerning specific pollutants.

There was consensus that the information in the preliminary assessment would be more effectively presented in a pollutant category format, similar to that of WHO publications. Since building design, maintenance, and ventilation cut across many pollutant categories, yet contribute unique concerns to indoor air pollution problems, it was also suggested that a separate chapter addressing these issues should be included. This would both emphasize the importance of building-type related concerns, and avoid the redundancy of repeating such concerns with each relevant pollutant. Still another general suggestion was that a crude exposure estimate for the various pollutants should be attempted, based on source prevalence and user patterns, but that the initial approach of attempting to quantify risk should be avoided. It was also recommended that summaries which clearly state the major knowns and unknowns should be given for each pollutant category.

ORD's response to these points has been to reformat the document into pollutant category form, addressing pollutant characterization, sources and occurrence, monitoring, exposure, health effects, and control and mitigation for each pollutant category. Welfare effects were organized into a separate chapter. Heating, ventilation and air-conditioning (HVAC) and building design and maintenance were also considered in a separate chapter. Finally, summaries were included for each pollutant category. These delineated the major knowns and unknowns for the category. In addition, a chapter was added which summarized information on a source-category basis. From a research and control viewpoint, it is clear that pollutants are emitted from sources, and that these sources must be mitigated in order to reduce pollutants which cause effects on health and welfare. Since the Research Needs Assessment, which discusses research options, is organized by source for practical and research purposes, the final chapter of the Information Assessment serves not only as summary, but leads into the source-category based Research needs portion.

More specific recommendations were to define clearly and to establish the distinction between monitoring and exposure, and to address the issue of total integrated exposure. Indoor air pollution is then to be put in the perspective of total exposure. The new version of the Information Assessment addresses these issues in the introduction.

Comments which were addressed to specific pollutants primarily addressed gaps in information, errors of fact, additional areas of concern, or differing views on approach to pollutants. Such concerns have been evaluated and the necessary changes have been made. Finally, an editing for consistency of style and to remove redundancies was suggested. Both a technical and stylistic editing have been done.

The primary issue in considering indoor air pollution is reducing risk to the public by reducing exposure. There have been a number of studies which calculated exposure to specific pollutants in a variety of indoor environments. Measurements were made under differing conditions of sampling methodology and averaging times, so that comparison of results across these studies is problematic. In further iterations of this document attempts to calculate meaningful exposures across the range of indoor pollutants will be made. In the present, a sketch of exposures derived from source prevalence information and work patterns, is provided, which does not attempt to incorporate the multiple assumptions and complex variables required for exposure calculation or modeling. However, since this is a vital issue in estimating risk, a special workshop addressing the various aspects of indoor air pollutant exposures is planned. Subsequent iterations of this document can then address this and the total exposure times. The report from Harvard detailing the findings from the workshop is attached.

SUMMARY OF PRINCIPAL FINDINGS FROM HARVARD WORKSHOP ON THE REVIEW OF THE PRELIMINARY ASSESSMENT DOCUMENT FOR THE U.S. EPA INDOOR AIR PROGRAMS

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EXECUTIVE SUMMARY

This report summarizes the principal findings from a workshop held at Harvard University on January 27-29, 1987 to review a preliminary assessment document for the EPA indoor air program. The purpose of the workshop was to evaluate the document for completeness and to recommend how EPA could best modify it bothe technically and structurally. A group of more than twenty scientists plus EPA and Harvard University staff participated in individual and general discussions to critique sections of the document and its overall format. Comments made both prior to and at the meeting have been provided to EPA separate from this report.

The purpose of this report is to summarize the essential comments and recommendations made during the meeting, it is not an independent assessment of the preliminary assessment document. Both general and pollutant-specific comments are presented. We have noted where there was not general agreement among the participants on certain issues. For example, the entire group of participants did not agree on how much or in what form risk assessment calculations should be handled in this document. However, most of the technical recommendations made by group leaders were acceptable to all the scientists attending the meeting.

The fore most recommendation, universally agreed upon, was to reformat the document, and recent WHO publications were suggested as a model. The reviewers also suggested that the document could be edited to eliminate repetitions, unnecessary details, and citations of secondary references. For certain pollutants, however, more information needs to be added to the document, especially on the distribution of population exposures and related technical issues (e.g., a clearer definition of terms and averaging times, health effects of peak and chronic exposures, and complex mixture/synergism problems).

Overall, the reviewers complimented the Agency on an excellent job of producing a complex and important document within a short period of time. Again, the major criticisms were directed towards reorganizing the existing material and presenting the technical information more coherently on the distribution of population exposures and health effects so that a priority ranking of indoor environment-related problems and mitigation alternatives could be implemented.

I. BACKGROUND

On January 27-29, 1987 Harvard University hosted a workshop to review a draft indoor air assessment document from EPA's Environmental Criteria Assessment Office (ECAO). Twenty scientists were invited to attend the workshop and a small group of Harvard staff served as facilitators and reporters. A panel of EPA staff also attended to observe the discussions. All of the workshop participants are listed in Table 1.

Before the workshop the non-EPA participants were asked to provide short written comments based on theri review of the document and respond to questions regarding the priority ranking of indoor pollutants. Copies of these materials were made available to EPA/ECAO.

Michael Berry and Harriet Ammann of EPA/ECAO opened the workshop by describing the purpose of the document, the reason it was generated, and the areas on which they would like the reviewers to concentrate.

The first part of the workshop, consisting of an afternoon and morning session, was devoted to small group discussions. Participants were divided into groups and given assigned topics. The leaders of the groups were asked to produce viewgraphs on what transpired in their group's discussions. The groups were arranged so that participants met with different colleagues in the two sessions. Table 2 shows the participation and topics covered in the small groups.

The second part of the workshop, also consisting of an afternoon and morning session, was devoted to plenary sessions where the leaders of the small groups presented viewgraphs on the topics their groups had discussed. The other participants could then comment on topics their groups had not covered. Copies of the viewgraphs presented in the plenary sessions were given to EPA/ECAO.

The comments provided below are meant as a summary of the major points that were made at the workshop. The first section deals with general comments on the assessment document. The second discusses points made about individual indoor pollutants.

II. OVERVIEW COMMENTS

This section draws upon the general discussions conducted in the plenary sessions as well as the reports from the two subgroups assigned to overview the document as a whole.

1. There is a need for an introductory section for the EPA Indoor Air Quality Assessment Document. This section would express the motivation behind the document and the need to examine indoor pollutants. This section should discuss the type of health effects that have been associated with indoor air pollutants. It should mention the number of people that are potentially exposed to indoor pollutants by concentrations or by sources. The section should outline the optional strategies that should be considered in reducing exposures, properly noting specific problems that different approaches might have. The last World Health Organization (WHO) indoor air quality report has a section on the advantages and disadvantages of various regulatory approaches.

2. There should be a section that discusses the physical and chemical properties of indoor air pollutants. Units of emission and concentrations could be explained in this section. Furthermore, the issues of averaging times and concentrations should be explained here as well. For some pollutants the long-term integrated concentrations are important, while for others the concern might be for short-term exposurew. Some indoor air pollutants have dynamic emission rates (e.g., formaldehyde, radon), for these pollutants the relationships between emissions and sampling times should be pointed out. This section is needed for two reasons: (1) to unify various units and terms, and (2) to establish the proper perspective for the reader to interpret data.

3. If the current structure is retained, the following changes are required:
    a. Define purpose of each chapter;
    b. Include a better concluding section for each chapter;
    c. Remove substantial redundancy;
    d. Revise mitigation section which is currently inadequate;

e. Include a section (not a chapter) on modeling. A comprehensive review of models, however, is not necessary. This section should state the usefulness and application of models to test theory, estimate parameters, simulate concentrations for study design or exposure estimation, and test mitigation strategies.

4. It s recommended that the report be shortened. A format similar to the WHO reports is suggested. Using this format, the references do not have to be comprehensive. Rather than using tables of data, the range of concentrations, emissions, and typical values would be reported.

5. The report should address integrated exposure assessment. However, Chapter 9 is not the proper approach. It was generally felt that Chapter 9 should be eliminated. Instead, the Agency should start the process of exposure assessment. While it may not be possible at this point to develop a full risk assessment with exposure and potency information on all indoor contaminants, the report should state what should be done and what is needed. The published risk numbers from other reports could be referred to with commentary about uncertainty.

The Agency should be heading towards an integrated exposure assessment that differentiates source contributions. It is a worthwhile goal to work towards the development of attributable risk concepts. For example, of all environmental factors affecting excess childhood respiratory illness, what percentage can e attributed to indoor versus outdoor sources? Similarly, from the population exposure to carcinogenic organic compounds, what is the attributable risk due to indoor exposures versus hazardous waste site exposures?

Risk assessment is an ongoing process that should be integral to EPA's Indoor Air Quality program and should be updated on a regular basis. Risk assessment could benefit EPA's indoor air quality program by preevaluating the usefulness of a particular research area and by testing mitigation strategies.

III. COMMENTS BY SPECIFIC POLLUTANTS

In this section we highlight the essential comments and recommendations made by workshop participants under each pollutant category. A significant fraction of these were discussed during the oral presentations of the group leaders at the workshop. Copies of transparencies listing most of these comments have already been provided to EPA/ECAO. In the following we summarize what we believe to be the most essential of these suggestions. It should be noted, however, that some of the comments listed below are more in the form of research recommendations rather than specific criticisms of the draft EPA document. Therefore, such topics may need to be acknowledged in the report instead of expending a great deal of effort at this time in assessing the present state of knowledge regarding such issues.

The following lists comments, criticisms, and recommendations by pollutant type directed at the document reviewed.

Radon

1. This section needs to be rewritten by an EPA expert on radon.
2. Welfare effects should be identified (i.e., impact on property values). It is expected that the needs of the real estate community (old and new homes) will become an important public issue. Furthermore, we need to address the new construction of houses quickly.
3. Health data need to be developed. This should include delineation of effects due to attached and unattached fraction.
4. Epidemiological studies are needed to evaluate the effects of chronic, low-level exposures to radon to assess mitigation strategies.
5. Inevitably, air cleaners will be used as a low-cost alternative to radon mitigation for secondary mitigation, and temporary mitigation. Also, air cleaners are becoming much more prevalent in homes today and their effect on health effects of radon must be known.
6. Research needs that should be emphasized also include:
    - Interpretation of seasonal and structural factors affecting radon concentrations;
    - Dose/response relationships;
    - Complex mixture interactions;

- Interpretation of recent epidemiology and new animal studies that are being published or underway; and
- Available mitigation options.

ETS

1. The report needs discussion of:
    - The specific compounds of interest;
    - Space/time considerations;
    - Identification of surrogates;
    - Bio-markers;
    - Questionnaires; and
    - Measurement approaches.
2. Temporal and spatial variability of exposures is an information gap that needs to be addressed.
3. Breakdown of exposure data by location and time.
4. Better characterization of:
    - Differences in MS/SS/ETS;
    - Interactions;
    - Decay and fate of contaminants;
    - Health effects on children; and
    - Adequacy of air cleaners and restrictions in public access buildings.

Nitrogen Dioxide

1. Multiple sources need to be mentioned.
2. Appliance misuse and improper venting of furnace, heaters, water heaters, and dryers may be substantial contributor to higher indoor NO_2 concentrations.
3. Measurement and better definition of short-term and peak NO_2 exposures should be undertaken.
4. Figure 5.1 should be deleted (dated material).
5. All combustion appliances should not be lumped together.
6. Because of increased use of kerosene heaters, the health effects of SO_2 and other byproducts of kerosene heaters need to be addressed.
7. Use of LA data should be cleared with Phil Baker of SoCal Gas.
8. Boston data are not available to public and should not be cited. Instead, the document could use Portage, WI data or Watertown, MA data as enclosed.

9. Topics for further research and discussion include:
   - NO_2 adsorption effects (fate and reaction products not well known);
   - Averaging time for health effects of NO_2 and dose-response data;
   - Better characterization of tails of the distribution of population exposures to NO_2;
   - Collecting field emission data on NO_2 source strengths for space and kerosene heaters; and
   - Evaluation of various mitigation options.

Carbon Monoxide

1. The document should mention that although CO is the only indoor pollutant that is known to be fatal, it is not considered to be a critical indoor health problem.
2. The document should mention the issue of concerns with the backdrafting of exhaust flue gases into homes resulting in elevated CO levels in homes but also in high concentrations of other combustion products as well.
3. The document should recognize the fact that a number of indoor chronic CO poisoning can go undetected due to a specificity of the symptoms. However, it is not likely that the small number of homes having CO problems can easily be detected even in large-scale monitoring studies.

Carbon Dioxide

1. CO_2 should be added as a separate section or under the HVAC section.
2. The document should include a discussion of the physical and chemical factors affecting CO_2 concentrations (ventilation, infiltration, transportation, and sinks).
3. It also should note the importance of building factors: design, operation, and maintenance monitoring.

Particles and Fibers

1. There is no need to redo what is in criteria documents.
2. The section on particles has technical errors and omissions. It is suggested that it should be structured along the WHO air quality guidelines document or the document on asbestos.
3. EPA should also utilize other Agency sources (e.g., WHO, NCRP, ASTM, DOE) in summarizing important data on exposures and health effects in comparable format.

4. Guidelines on air cleaners or a program to define the value of these devices should be included.
5. Man-made-mineral fibers need to be included in the document.
6. The EPA asbestos program needs careful evaluation. Problems will occur when it is recognized that home exposures may be very large following asbestos removal.

Biologicals

1. The document needs to indicate that aeropathogens and aeroallergens are major contributing cause of acute morbidity.
2. Terms and sampling systems should be defined.
3. The major needs in this field are for:
   - Baseline data on concentrations and health effects of these pollutants;
   - Standardization of measurement methods;
   - Clear definition of humidifier fever and other allergic illnesses;
   - Mycotoxin and endotoxin health effects and their carcinogenic potential (use of antimicrobials may have unwanted side effects); and
   - Education of public, building designers, engineers, and supervisors regarding reservoirs, amplifiers, and disseminators of these pollutants.

VOCs

1. Information contained in Table 3-6 should be synthesized better (give concentration values, not just references).
2. The difference between monitoring and exposure should be clarified.
3. The difficulties with "unknown" VOCs and the heterogeneous chemical composition of VOCs should be mentioned.
4. Information on averaging times for the data presented should be provided.
5. Review of sources of VOCs should include outside sources as well as commuting exposures. Automobile emissions should be included as important sources of outdoor as well as indoor organic pollutant exposures.
6. A table should be provided to show an overview of emitted organics by source type.

7. The possibility of biological markers and breath samples as indicators of exposure needs to be mentioned.
8. The qualitative character of Table 6-16 should be stressed. Remove "use" column from this table.
9. A new section should be written to discuss modeling.
10. Areas where scientific knowledge is insufficient should be identified, such as:
    - Source emission characterization;
    - Acute and chronic health effects;
    - Odor and sick building syndrome problems;
    - Toxicology of mixes;
    - Better instrumentation and/or surveys; and
    - Effects of temperature, humidity, and ventilation.
11. Mitigation or control considerations should be emphasized: source removal, consumer education, and product use, substitution

Formaldehyde

1. The document should refer to other source documents since scientific knowledge on formaldehyde sources and exposures even effects is quite good.
2. A reference to the Canadian work should be added in Table 6-13 and footnotes to Table 6-12.

Pesticides

1. Section is disjointed as now written. The knowledge of pesticides is in general adequate, but the chapter is not.
2. The document should summarize and reference available documents.
3. The document should give some guidance of where exposures may be the most important (offices, restaurants, etc.).
4. Information on biological markers should be included in the exposure section.
5. Section 6.12 is inadequate and should be rewritten.
6. Occupational data on toxicity should be included so that extrapolations for the general population can be made.

7. A summary of observed health effects should be included. If different effects in various groups, such as infants, are expected, they should be noted.
8. A summary of available health effects data at different pesticide concentrations should be presented.

IV. RECOMMENDATIONS

The reviewers of the document all recognized the challenge faced by EPA in putting together an ambitious document within a short period of time. Although the document is quite extensive in its coverage, it suffers from "multiple-author syndrome." The most important suggestion made by the reviewers was the need to restructure the document to avoid repetition and the superfluity of details that could be found elsewhere. The reviewers thought the document needed an introductory section discussing the purpose and key issues of concern in terms of population exposures, potential health effects, and available control strategies. Also suggested was the possibility of considering an alternative format similar to those used in recent WHO reports.

Regarding the pollutant-specific sections, there were a number of recommendations for displaying exposure and effect information more clearly. Issues of consistency in units, terminology, synergisms, and measurement methods were also raised. The need for better characterizing time issues and peak exposure effects was also pointed out (VOC, radon, NO_2). For certain pollutants (particles and formaldehyde) briefer presentations supported with primary references or more complete information sources were suggested. However, for the pesticide and radon sections additional information on exposures and health effects by population groups were recommended.

Another suggestion was to include a new or a separate section on modeling or building systems that includes modeling, ventilation, welfare, comfort, energy, and other related issues. Adding a discussion on indoor CO_2 and its relationship to HVAC systems was also considered by many reviewers to be worthwhile.

The participants agreed that a more complete presentation of the distribution of population exposures to various pollutants would be useful. Recommended in particular was a graphical display of concentrations or exposures plotted against the number of people exposed in order to aid the evaluation of the extent of the indoor air problem. However, there was no consensus among the workshop participants as to whether the Agency should carry out the next logical step, namely, to perform a population-based risk assessment using the exposure distributions and the health-risk potencies.

One group was reluctant to consider a (risk-based) ranking methodology for pollutants. They thought that no present methodology would allow an assessment that would yield meaningful results, that is, within reasonable confidence/error limits. Another group, however, suggested putting bounds on the estimates of the number of populations at risk and identifying control alternatives by examining the tails of the distributions (for exposures as well as for percentage of population affected) and comparing these to the total exposures or risks. Consideration of error and uncertainty of estimates (though formally complex to implement) was also recommended.

It is clear that display of exposures by population groups or by region would be helpful in identifying the magnitude of a certain indoor air pollution problem. Although a proper risk assessment application should be among one of the key goals of an indoor air research program, it could lead to serious controversy if hastily or incorrectly done. As we mentioned above, we suggest a progressive effort in this area, rather than calculating numerical mortality or morbidity risk estimates without first developing a basic framework for risk assessment. Qualitative ranking of information, exposures, potential risks, and priorities for various mitigation options should perhaps be the starting point. However, while the information bases on exposures and effects are being developed, the Agency should also investigate how best to quantify the nature and magnitude of health risks resulting from exposure to pollutants of both indoor and outdoor origin.

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TABLE 2. GROUP ASSIGNMENTS AND RESPONSIBILITIES ON JANUARY 27, 1987

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McCann	Lippmann	Yocum	Stolwijk	Molhave
Grimsrud	Rand	Burge	Letz	Smith
Benowitz	Turner	Broder	Morey	Walkinshaw
Harvard University Participants				
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EPA Observers				
Graham	Lewtas	Sparks	Wallace	No EPA
Tucker	Rodes	Otto	Tichenor	
Elias	Costa	Repace	Kawecki	
		van Hook		
Focus of Review				
Combustion Products- CO, NO_2	Particles	Formaldehyde	Pesticides	Document Overview
Pesticides	Combustion Products- CO, NO_2	Particles	Combustion Products- CO, NO_2	
Particles	Formaldehyde	Pesticides	Formaldehyde	

TABLE 2. GROUP ASSIGNMENTS AND RESPONSIBILITIES ON JANUARY 28, 1987

Group Leaders and Members				
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Stolwijk	Walkinshaw	Broder	Letz	Grimsrud
Harvard University Participants				
Brunekreef	McCarthy	Ryan	Yanagisawa	Rudnick
EPA Observers				
No EPA	Lewtas	Costa	Tucker	van Hook
	Repace	Graham	Wallace	Tichenor
	Rodes	Sparks	Otto	Elias
	Kawecki			
Focus of Review				
Document Overview	ETS	Biological	Organics	Radon
Summary of Questions Responses	Organics	ETS	Radon	Biological
	Radon	Organics	Biological	ETS